A STUDY OF IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION

Dissertation submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations

for the award of the degree of

M.D. GENERAL MEDICINE (BRANCH - I)

INSTITUTE OF INTERNAL MEDICINE

MADRAS MEDICAL COLLEGE

CHENNAI 600 003

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2015

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION"** is a bonafide work done by **DR.RAGAVAN K**, Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, during March 2014 to August 2014 in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I General Medicine, under our guidance and supervision, during the academic year 2012 - 2015.

Prof. S.TITO, M.D.,
Director i/c & Professor,
Institute of Internal Medicine,
MMC & RGGGH,
Chennai - 600003

Prof. R.PENCHALAIAH, M.D.,
Professor of Medicine,
Institute of Internal Medicine,
MMC &RGGGH,
Chennai - 600003

Prof.R.VIMALA, M.D.,

Dean,
Madras Medical College &
Rajiv Gandhi Government General Hospital
Chennai- 600003

DECLARATION

I, **Dr. RAGAVAN K** solemnly declare that dissertation titled **"A STUDY OF IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION"** is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during March 2014 to August 2014 under the guidance and supervision of my unit chief **Prof. R.PENCHALAIAH, M.D.,** Professor of Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine**

Place : Chennai **(Dr.RAGAVAN K)**

Date :

ACKNOWLEDGEMENT

I owe my thanks to Dean, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 **Prof.R.VIMALA, M.D.,** for allowing me to avail the facilities needed for my dissertation work.

I am grateful to beloved mentor **Prof.Dr.S.TITO, M.D.,** Director i/c and Professor, Institute of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai for permitting me to do the study and for his encouragement.

I am indebted to my chief **Prof.R.PENCHALAIAH, M.D.,** Professor, Institute of Internal Medicine for his guidance during this study.

I am extremely thankful to **Prof.Dr.M.S.RAVI, M.D., D.M.,** Professor and Head of the Department of Cardiology for guiding me and allowing me to use the departmental facilities.

I am extremely thankful to my Assistant Professors **Dr.M.Sharmila, M.D.,** and **Dr.S.Aparna, M.D.,** for their guidance and encouragement.

I am also thankful to all my unit colleagues for their full cooperation in this study and my sincere thanks to all the patients and their families who co-operated for this study. Finally I thank my parents and all my family members who gave me their full support and co-operation in completing the dissertation.

CONTENTS

“A STUDY OF IMPACT OF ADMISSION MEAN PLATELET ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION”

ABSTRACT:

BACKGROUND AND AIMS:

The role of platelets in pathogenesis of myocardial infarction is well established. Larger metabolically and enzymatically platelets are released during atherosclerotic plaque rupture. Reactivity and size of the platelets are measured by mean platelet volume and it can be related to the burden of thrombus measured by post thrombolysis TIMI flow.

METHODS:

Data from Institute of internal medicine and Department of cardiology, Madras medical college, Rajiv Gandhi Government General Hospital were analysed by measuring mean platelet volume on presentation and its relation to post thrombolysis TIMI flow. Patients were divided into two groups having mean platelet volume 9.5 as target. Evaluated by Pearson chi square test.

RESULTS:

In our study out of 40 patients most of the patients were in the age group of 41-60 years(27 patients) and in sex distribution males were in large number (27 patients).Significant correlation obtained between mean platelet volume and

infarct size, TIMI flow ,platelet count ,left ventricular systolic function, number of vessels involved.

CONCLUSIONS:

Successfulness of thrombolysis was inversely proportional to admission mean platelet volume. Infarct related artery patency and TIMI flow were inversely proportional to admission mean platelet volume. Platelet count was inversely proportional to admission MPV. Total count has correlated with infarct size.

Key words: Myocardial infarction, TIMI flow, mean platelet volume

INTRODUCTION

Myocardial infarction continues to major health problem both in industrialized world and in developing countries, even after advances in diagnosis and management. Mortality from STEMI has declined steadily. Decrease in mortality is attributed to fall in incidence of STEMI and fall in the case fatality rate. Nearly 10% of myocardial infarcts occur in people under age 40, and 45%occur in people under age 65. Blacks and whites are equally affected. Throughout life, men are at significantly greater risk than women.

Management of STEMI has progressed through various phases. In the first half of 20th century was “CLINICAL OBSERVATION PHASE” in which detailed recording of physical and laboratory findings with little active treatment for the infarction.

In mid 1960s coronary care unit phase begins and detailed analysis of cardiac arrhythmias. After the introduction of pulmonary artery floatation catheters the stage of high technology phase started. The modern reperfusion era was occupied by intracoronary and intravenous fibrinolysis.

The dominant cause of the IHD syndromes is insufficient coronary perfusion relative to myocardial demand, due to chronic, progressive atherosclerotic narrowing of the epicardial coronary arteries, and variable degrees of superimposed acute plaque change, thrombosis, and vasospasm.

The role of platelets in thrombus formation is already well studied. At

the time of myocardial infarction, new large sized platelets are released from the bone marrow. The large sized platelets show high mean platelet volume. Hence the admission MPV has been shown to be a strong and independent predictor of impaired angiographic reperfusion in acute ST-segment elevation myocardial infarction. Reestablishment of IRA flow is associated with decreased mortality in patients with acute myocardial infarction. It may be speculated that failure to restore epicardial coronary blood flow after thrombolytic administration could contribute at least in part to higher morbidity and mortality rates in patients with an elevated MPV.

AIMS AND OBJECTIVES

AIMS & OBJECTIVES

- To study about the impact of admission mean platelet volume on the efficacy of thrombolysis in ST elevation myocardial infarction.

- To study about the impact of admission mean platelet volume and successfulness of thrombolysis, left ventricular function and coronary patency after thrombolytic therapy in ST elevation myocardial infarction

REVIEW OF LITERATURE

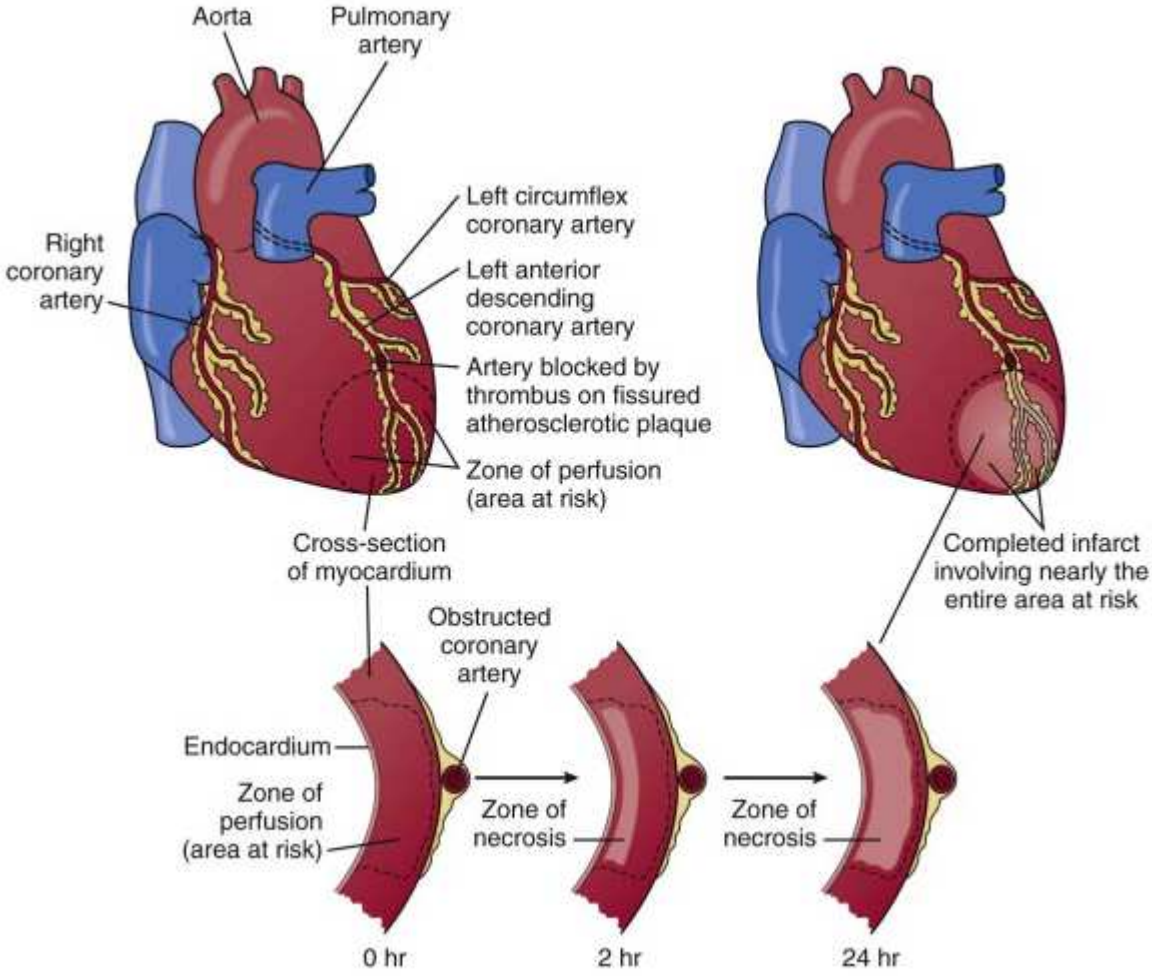
REVIEW OF LITERATURE

MYOCARDIAL INFARCTION:

Defined pathologically by myocardial cell death by prolonged ischemia. Clinical diagnosis requires integrated biochemical, electrocardiographic and imaging .[1]

Classification of Myocardial infarction:[1,2]

TYPE	FEATURES
1	Spontaneous due to plaque rupture, erosion, fissuring or dissection
2	Demand supply mismatch (anemia, hypotension, hypertension, coronary spasm, coronary embolism)
3	Sudden unexpected cardiac death(new ST elevation, new LBBB, Angiography evidence)
4a	PCI associated
4b	Stent thrombosis
5	CABG associated



Pathophysiology:[3,4]

Types of abnormal contraction patterns:

- Hypokinesia.
- Akinesia
- Dyssynchrony
- Dyskinesia.

Ischemia at distance:[5]

Collaterals loss due to infarct artery

Contractile dysfunction in non-infarcted zone

HEART MUSCLE:

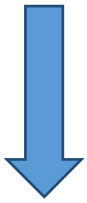
Gross pathology:[6]

Difficult to identify within 6 hours.

1) Transmural infarction
2) Non transmural (Sub-endocardial infarction)

Transmural infarction:

Occlusion of coronary artery

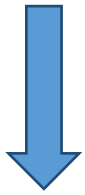


Full thickness myocardial necrosis

Single coronary artery localization

Nontransmural myocardialinfarction :

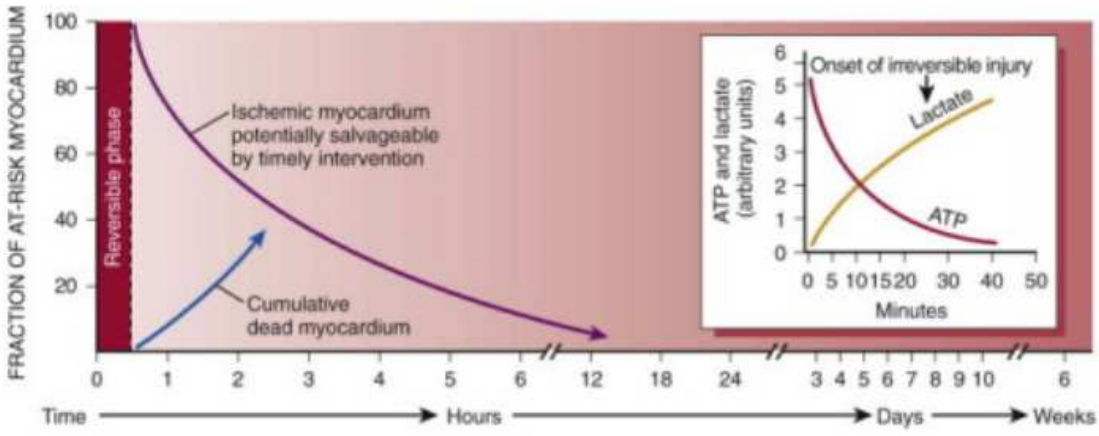
Severely narrowed but patent coronary arteries



Nontransmural patchy infarction

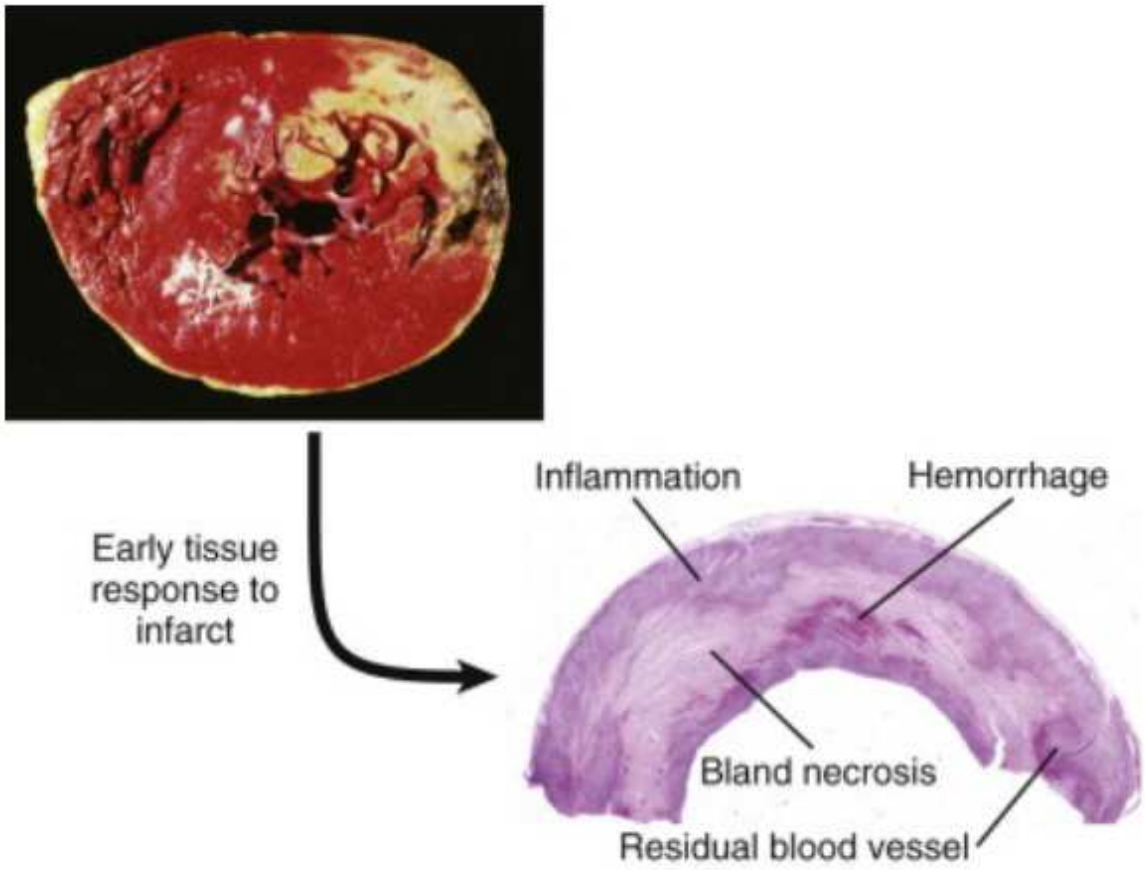
HISTOLOGIC AND ULTRASTRUCTURAL CHANGES:[7,8,9]

TIME	GROSS	LIGHT MICROSCOPY	ELECTRON MICROSCOPY	TTC DEFECT
0-3hrs		Waviness of border cardiac fibres	Depletion of glycogen, myofibrillar relaxation, swelling of mitochondria	Present
3-12hrs		Coagulation necrosis, infiltrate of neutrophils, edema	Disruption of sarcolemma, amorphous densities in mitochondria	Present
12-24 hrs	Pallor	Coagulation necrosis plus contraction bands		Present
1 day – 10 days	Pallor plus periphery hyperemia	Myofibre disintegration, macrophage phagocytosis		Present
10 – 6 weeks	Soft and yellow	Phagocytosis completed, neovascularised granulation tissue		Present



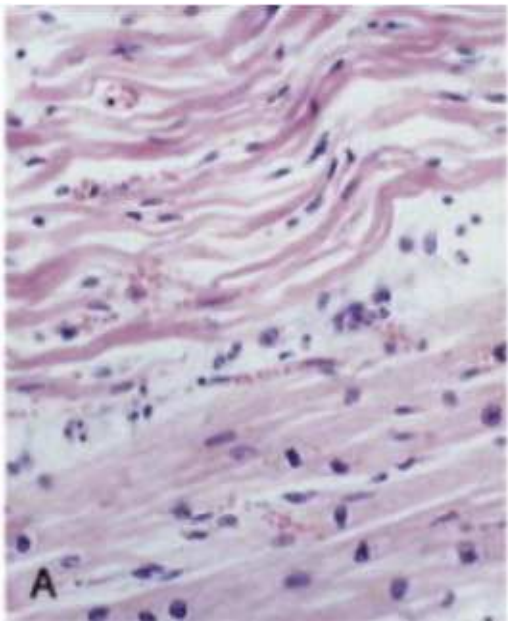
TTC (triphenyltetrazoliumchloride) staining[3]

- Intact, non infarcted myocardium + active LDH → brick red colour
- Infarcted myocardium + inactive LDH → unstained pale zone



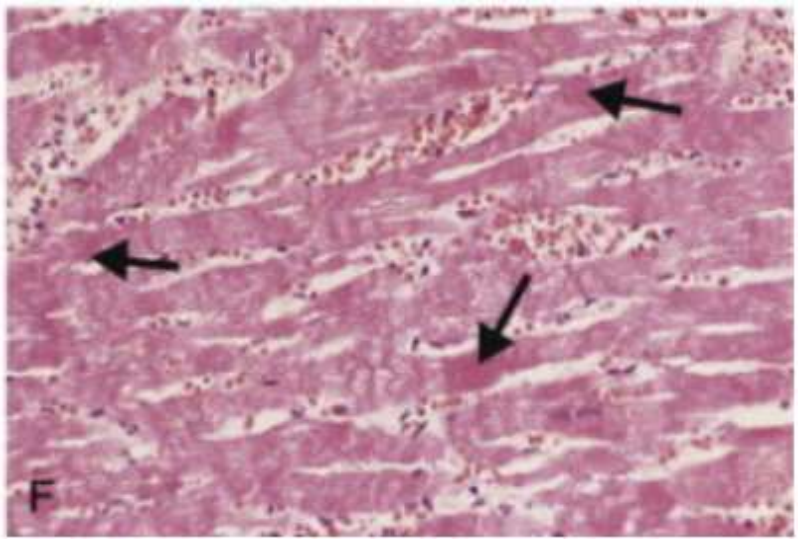
COAGULATION NECROSIS:

Central region persistent ischemia leads to muscle cells arrest in relaxed state with mitochondrial densities.



CONTRACTION BANDS/COAGULATIVE MYOCYTOLYSIS:[10]

Reflow following severe ischemia leads to calcium influx into periphery of infarcts leads to arrest of cells in the contracted state.



MODIFICATION OF PATHOLOGIC CHANGES BY REPERFUSION:[11]

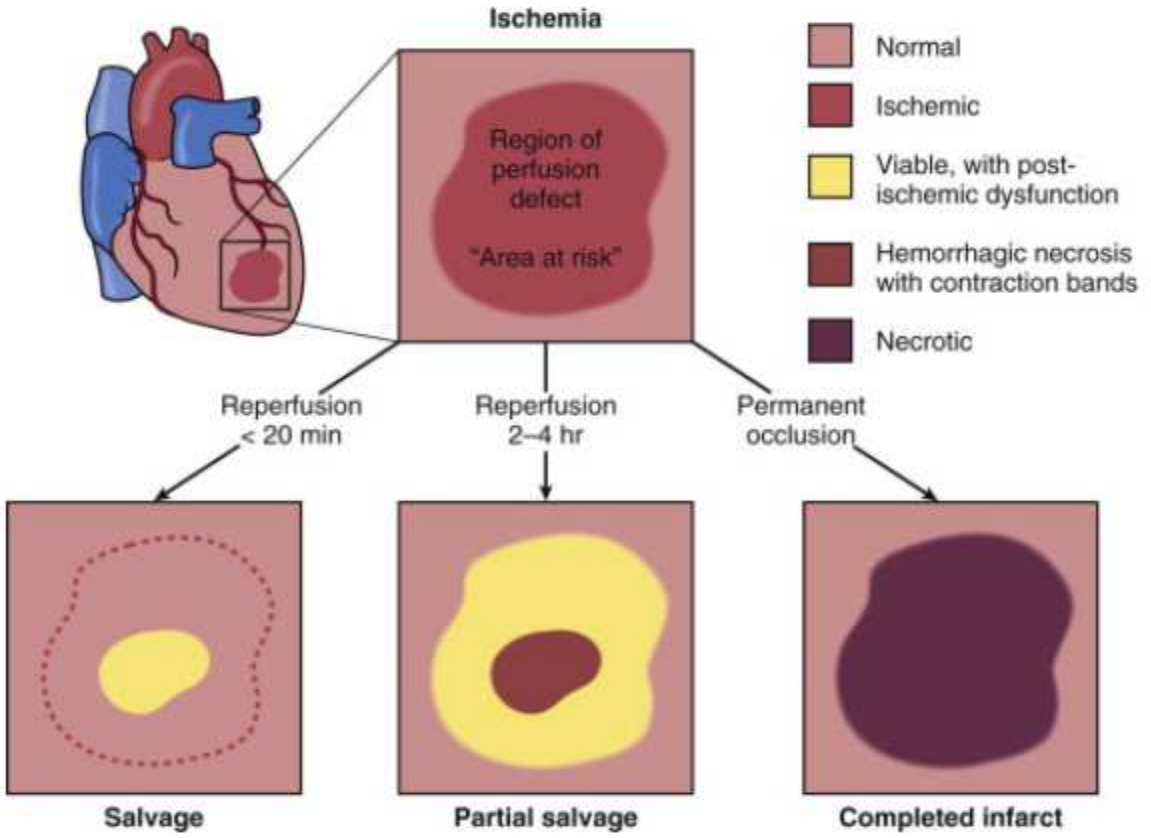
Early reperfusion <20 minutes—Prevents necrosis

Area of necrosis is directly related to total coronary artery occlusion time, oxygen consumed by myocardium, collateral blood flow.

Reperfusion of infarcted myocardium produces early and exaggerated peaking of CK-MB, and Troponin I & T due to accelerated wash out of intracellular protein.

RIGHT VENTRICULAR INFARCTION:[12]

- Occurs in 50% of patients with inferior wall infarction.
- Isolated RVMI 3—5% of autopsy proven cases
- Long ischemic periods sustainability and excellent contractile function recovery.



ATRIAL INFARCTION:[13]

10% of cases

Most common site—Right atrial appendage

Frequent atrial arrhythmias.

COLLATERAL CIRCULATION:[14]

Collateral circulation well developed in following conditions

>75% stenosis in one or more arteries

Severe anemia, COPD, cyanotic congenital heart disease

Hypertrophy of left ventricle

Collaterals flow is inversely proportional to infarct size and aneurysm.

PATHOPHYSIOLOGY:

LEFT VENTRICULAR FUNCTION:[15]

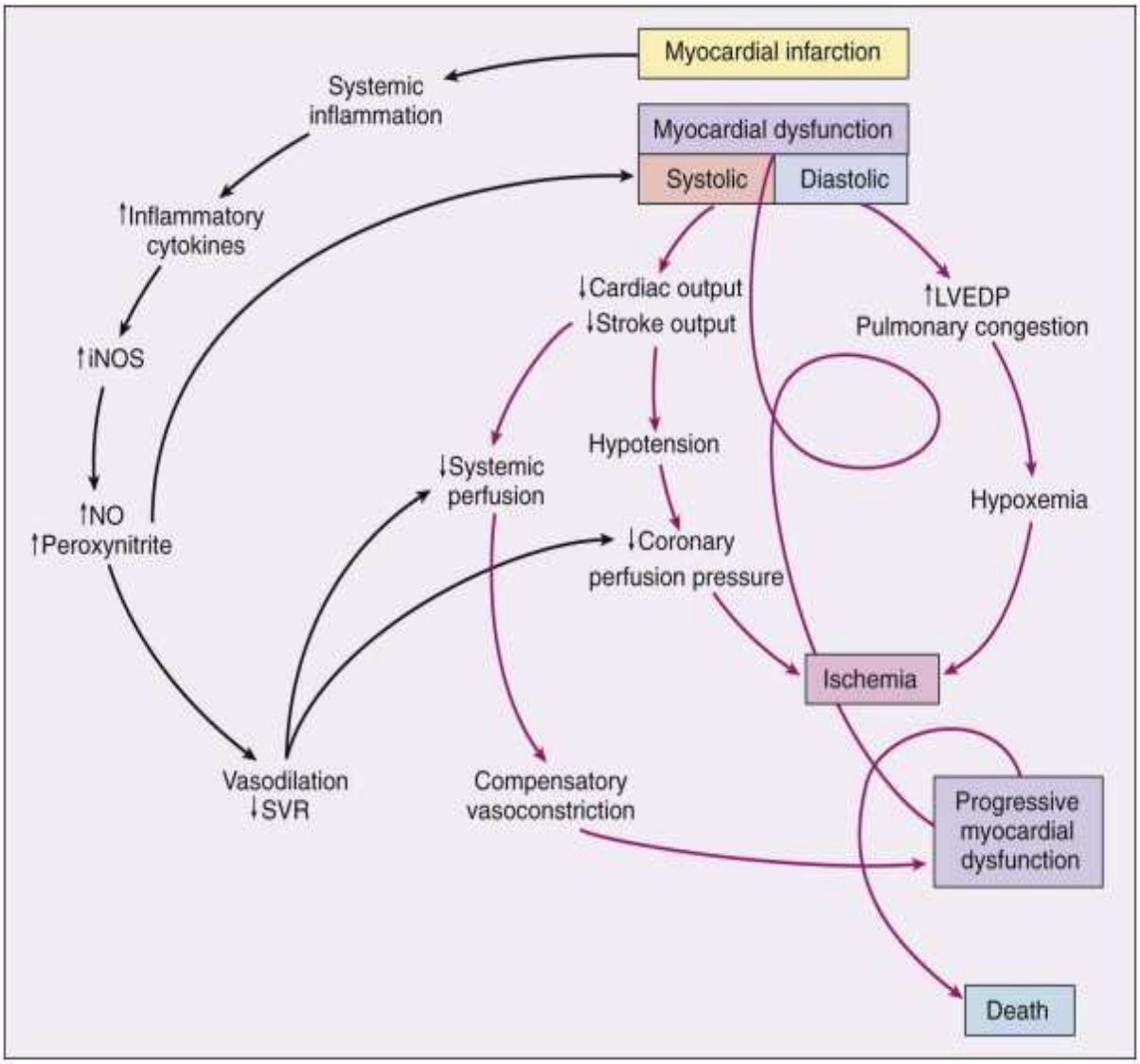
Sufficient ischemic injury

Left ventricular pump failure

Decreased cardiac output, decreased stroke volume, decreased blood pressure, increased end systolic volume

Hemodynamic predictor of mortality is rise in end systolic volume.

Circulatory mechanism in MI[16]



REMODELLING[17]:

Changes in size, shape, thickness of infarcted and noninfarcted segments.

Infarction of myocardium

↓

Hypokinesia or akinesia

↓

Sympathetic system activity

Frank-Starling mechanism

↓

Dyskinesia of noninfarcted region

Paradoxical systolic bulging

↓

Thinning and elongation of infarct segment

Abnormal contracting segment>15%--Ejection fraction decreases[18]

Abnormal contracting segment>25%--Clinical heart failure

Abnormal contracting segment>40%--Cardiogenic shock

INFARCT EXPANSION[19]:

Dilation and thinning which is acute otherwise not explained by additional myocardial necrosis.

Determinants[19]:

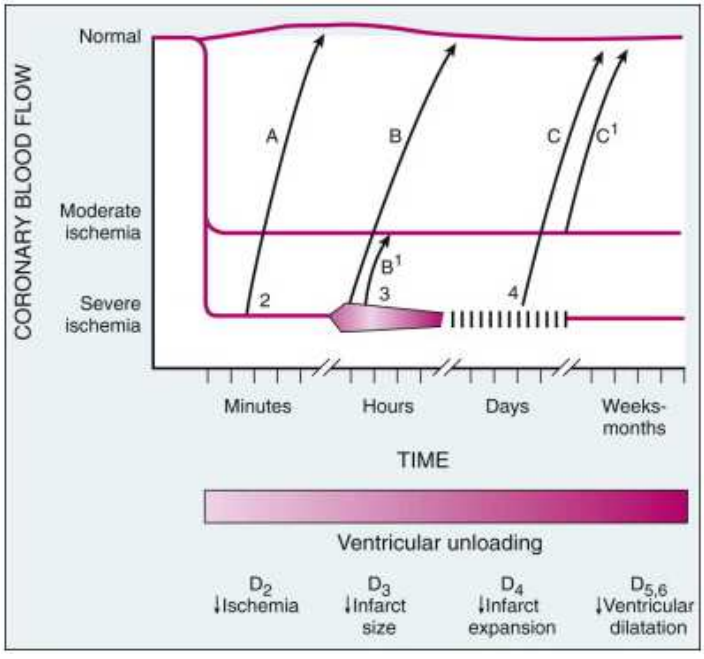
1. Muscle bundle slippage
2. Myocardial cell disruption
3. Necrotic zone tissue loss

Preinfarction wall thickness inversely proportional to infarct thinning.

- Apex –Thinnest region highly vulnerable to remodeling.

ECHO : Non contractile region elongation

EFFECTS OF TREATMENT:



20

[20]Inhibitors of RAAS	-	Decreases remodeling
Angiotensin II blockade	-	Dysfunction of endothelium attenuated.
		Antiatherogenesis
Aldosterone blockade	-	Decrease in collagen deposition
		Decrease in ventricular arrhythmias

CLINICAL FEATURES:

General appearence[21]

- Anxious
- Restless
- LEVINE SIGN –Clenched fist held against chest
- Skin pallor and cold perspiration—Sympathetic stimulation and left ventricular failure.
- Pink, frothy, blood stained sputum-Pulmonary edema
- Cold clammy skin, cyanosis, shock-cardiogenic shock.

Circardin periodicity:[22,23]

Early morning hours

↓

Increased catecholamines,cortisol

Increased platelet aggregability

↓

Peak hours 6am –NOON

Periodicity was absent in patients receiving β blocker and aspirin

Nature of the pain:[24]

- Duration – 30minutes to hours
- Character – Compressing,choking,oppressing,sensation of heavy weight, squeezing
- Site – Retrosternal, substernal
- Radiation—Left shoulder, left ulnar aspect of arm,wrist and little finger, also radiates to neck, jaw, interscapular region

Pain produced by injured or ischemic myocardium not from necrotic tissue.

Associated symptoms[24]:

- Vagal reflex or Bezold –Jarisch reflex

  ↓

  Nausea, vomiting

- Breathlessness
- Palpitation
- Sense of impending doom
- Cold perspiration
- Silent MI—Diabetes mellitus, Hypertension

PHYSICAL EXAMINATION:[25]

Heart rate:

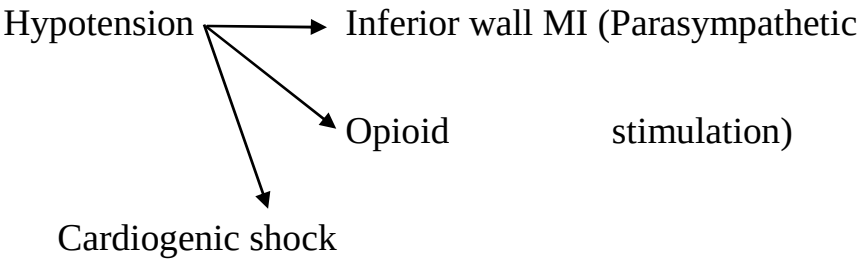
Bradycardia –Inferior or right ventricular MI

Tachycardia –Anterior wall MI

Blood pressure:[26]

Normotension –Non complicated MI

Hypertension –Early hours due to adrenergic discharge



Temperature:

Begin to rise within 4 to 8 hours

Rectal temperature –38.3°c to 38.9°c

Temperature normalizes within 4—5 days

Respiration:[27]

Rate –Slight elevation and settles with treatment

>40/min –Pulmonary edema

Character –Cheyne-Stokes in cardiogenic shock, elderly, heart failure, opioid

Jugular venous pulse:[26]

Most of the patients not elevated

Prominent ‘a’ wave – LV failure induced pulmonary hypertension

Tall ‘cv’ wave – right ventricular papillary muscle ischemia induced tricuspid regurgitation

Carotid pulse:

Pulsus alternans – severe left ventricular dysfunction

Small pulse – reduced stroke volume

Sharp, brief upstroke – left to right shunt due to ventricular septal rupture or mitral regurgitation

CHEST EXAMINATION:

Killip and Kimball classification [28,31]

- Class I – no rales, no S3
- Class II – rales <50% lung fields, +/- S3
- Class III – rales >50% lung fields, and pulmonary edema
- Class IV – cardiogenic shock

CARDIAC EXAMINATION:[29,30]

Palpation:

Presystolic pulsation – left atrial vigorous contraction against non-compliant ventricle.

An outward movement of the left ventricle in early diastole coinciding with S3 represents systolic dysfunction of left ventricle.

Heart sounds:

- S1 – soft and muffled represents prolonged PR interval
- Paradoxical splitting of S2 – left bundle branch block
- S3
    - severe LV dysfunction due to elevated filling pressures
    - Can also occur in mitral regurgitation and ventricular septal rupture
- S4 almost universally present in STEMI patients in sinus rhythm and has no prognostic value

Murmurs:

- Mitral valve apparatus dysfunction (papillary muscle dysfunction or dilatation of LV) – produces transient systolic murmur

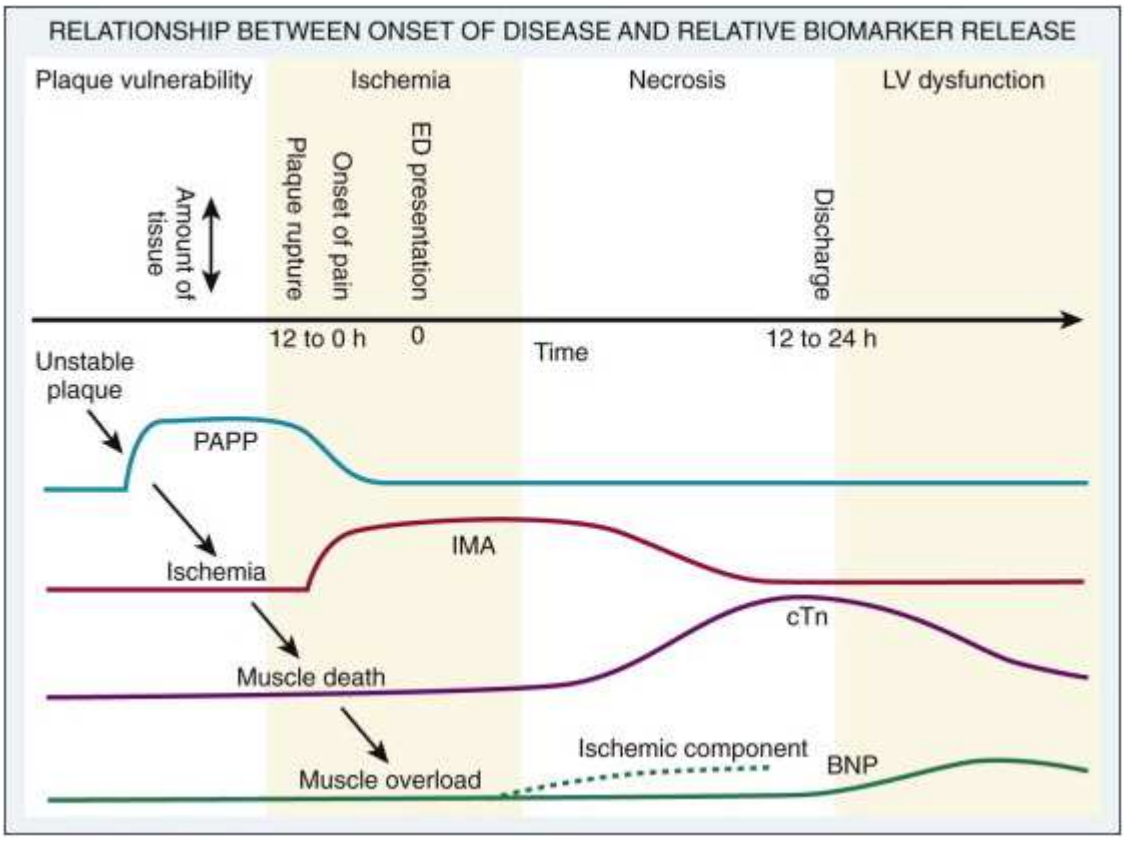
- Papillary muscle rupture – apical holosystolic murmur with thrill
- Ventricular septal rupture – holosystolic murmur with thrill along left sternal border
- Right ventricular failure causing tricuspid regurgitation – systolic murmur along left sternal border with positive Carvallo's sign
- Friction rub – heard within one day or as late as two weeks, most common on second or third day. Highly evanescent finding.
- Dressler's syndrome – late onset pericarditis (even after 3 months)

LABORATORY FINDINGS:[32,33,34]

Serum markers of myocardial damage:

Necrosis of myocardium → sarcolemmal membrane disruption → diffusion of intracellular macromolecules in to the cardiac interstitium → further diffusion in to micro vasculature and lymphatics.

PAPP released in to the blood during the stage of plague vulnerability. After plaque rupture IMA is released during the initial period of ischemia.



Creatine kinase isoenzymes:

3 isoenzymes

1. MM – skeletal muscle
2. BB – brain and kidney
3. MB – heart (small quantity is seen in small intestine, diaphragm, prostate, tongue, uterus)

Relative index of CK-MB:

$\text{CK MB MASS}/\text{CK ACTIVITY}$ > 2.5 → cardiac origin

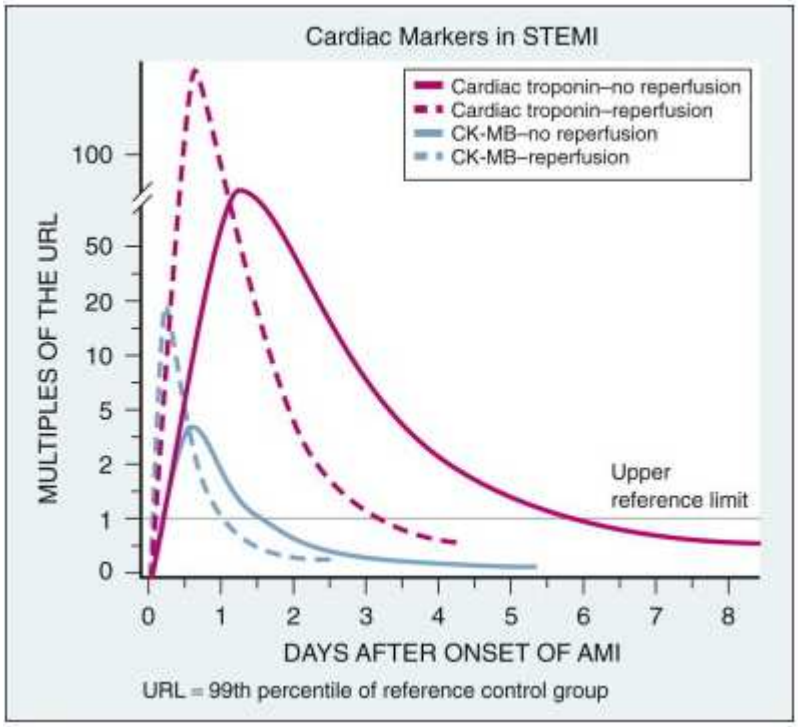
False positive results can occur in myocarditis, cardiac catheterization, shock, cardiac surgery, trauma and skeletal muscle injury.

Cardiac specific troponins:

Troponin complex

- Troponin C – binds to calcium
- Troponin I – binds to actin and inhibits actin-myosin interaction, 2-3% in cytosol
- Troponin T – binds to tropomyosin, 6% in cytosol

Cut off value – Value exceeding 99% of the reference control group



Biomarkers for Evaluation of Patients with ST-Segment Elevation Myocardial Infarction[32,33,34]

BIOMARKER	MOLECULAR WEIGHT (DA)	RANGE OF TIME TO INITIAL ELEVATION (HR)	MEAN TIME TO PEAK ELEVATIONS (NONREPERFUSED)	TIME TO RETURN TO NORMAL RANGE
Frequently Used in Clinical Practice				
MB-CK[*]	86,000	3-12	24 hr	48-72 hr
cTnI[†]	23,500	3-12	24 hr	5-10 days
cTnT	33,000	3-12	12 hr-2 days	5-14 days
Infrequently Used in Clinical Practice				
Myoglobin	17,800	1-4	6-7 hr	24 hr
MB-CK tissue isoform	86,000	2-6	18 hr	Unknown
MM-CK tissue isoform	86,000	1-6	12 hr	38 hr

Classification of Different Types of Myocardial Infarction: Suggested Grid for Reporting Results

MULTIPLES X 99%	MI Type					TOTAL NO.
	1 (SPONTANEOUS)	2 (SECONDARY)	3(SUDDEN DEATH)	4 (PCI)	5(CABG)	
1-2^{x}						
2-3^{x}						
3-5^{x}						
5-10^{x}						
>10^{x}						
Total no.						

OTHER TESTS:

Lipid profile:

Total cholesterol and HDL remain at baseline during the initial 24-48 hours, after that both values begin to fall HDL > Total cholesterol

In patients admitted after 48 hours, measurement of serum lipids is advised after 8 weeks.

Hemostatic markers:

Leukocytes – elevated according to the level of necrotic process, directly proportional to in-hospital mortality, count ranges from 12,000 – 20,000, correlates with the angiographic appearance and clinical outcomes.

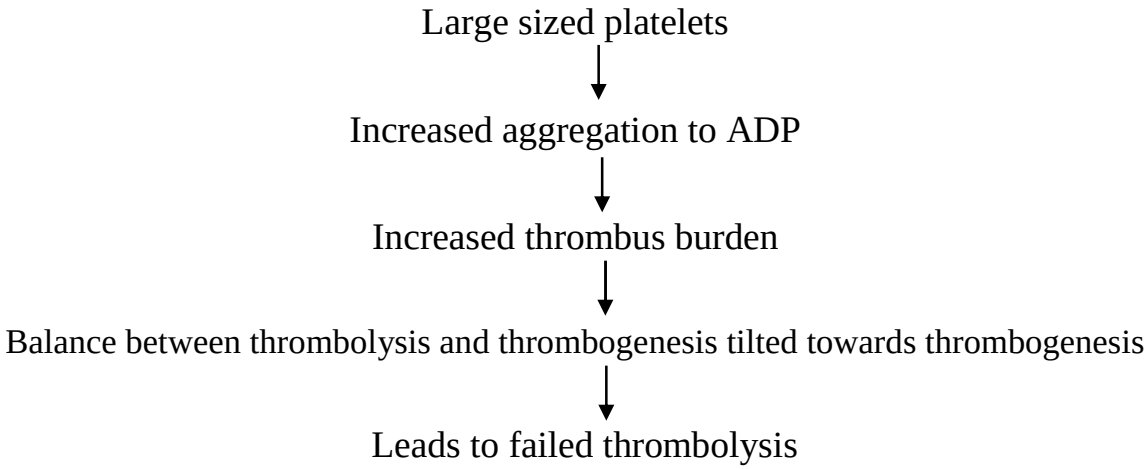
Blood viscosity – elevated due to hemoconcentration, elevated levels of alpha 2 globulin and fibrinogen

ESR – elevated due to increased level of fibrinogen not related to level of necrotic process

Hemoglobin – J shaped relationship with the clinical events, levels <14 leads to decreased oxygen supply, levels >17 leads to polycythemia and increase in blood viscosity

CRP – elevated and correlates with the infarct related artery and heart failure

Platelets – metabolically and enzymatically active platelets are released. They are large sized and contains increased granules, increased levels of thromboxane, beta thromboglobulin, P-selectin, GP IIb/IIIa receptor and fibrinogen. Platelet factor 4, thrombin, antithrombin complex and fibrinopeptide A are increased and correlates with mortality



Mean platelet volume:

Large sized reactive platelets are measured by mean platelet volume

Instrument used : Beckman – Coulter LH780 Hematology analyser

Normal MPV: 7.5 – 11.5 fL

In acute MI – MPV has significant correlation with the prognosis

Platelet count – at the initial hours of MI, platelet counts may be decreased due to consumption which correlates with the prognosis.

ELECTROCARDIOGRAM:[35]

In 1887 Waller directly recorded cardiac electrical potentials.

In 1901 Einthoven registered electrical activity by string galvanometer.

Principle:[36]

Ionic fluxes across cell membrane and cells produce transmembrane ionic currents which are synchronized by activation and recovery of cardiac cells generating electrical field in and around the heart

ESC STEMI DIAGNOSTIC CRITERIA:

SEX	ST ELEVATION	LEADS
MALE <40 years >40 years	 ≥0.25 mV ≥0.2 Mv	 atleast 2 contiguous leads
FEMALE	≥0.15 mV ≥0.1 mV	V2, V3 Other leads

POSITIONING OF LEADS:[37]

LEAD TYPE	POSITIVE INPUT	NEGATIVE INPUT
Standard Limb Leads		
Lead I	Left arm	Right arm
Lead II	Left leg	Right arm
Lead III	Left leg	Left arm
Augmented Limb Leads		
aVR	Right arm	Left arm plus left leg
aVL	Left arm	Right arm plus left leg
aVF	Left leg	Left arm plus right arm
Precordial Leads*		
V_1	Right sternal margin, fourth intercostal space	Wilson central terminal
V_2	Left sternal margin, fourth intercostal space	Wilson central terminal
V_3	Midway between V_2 and V_4	Wilson central terminal
V_4	Left midclavicular line, 5th intercostal space	Wilson central terminal
V_5	Left anterior axillary line [†]	Wilson central terminal
V_6	Left midaxillary line [†]	Wilson central terminal
V_7	Posterior axillary line [†]	Wilson central terminal
V_8	Posterior scapular line [†]	Wilson central terminal
V_9	Left border of spine [†]	Wilson central terminal

* The right-sided precordial leads V_3R to V_6R are taken in mirror image positions on the right side of the chest.

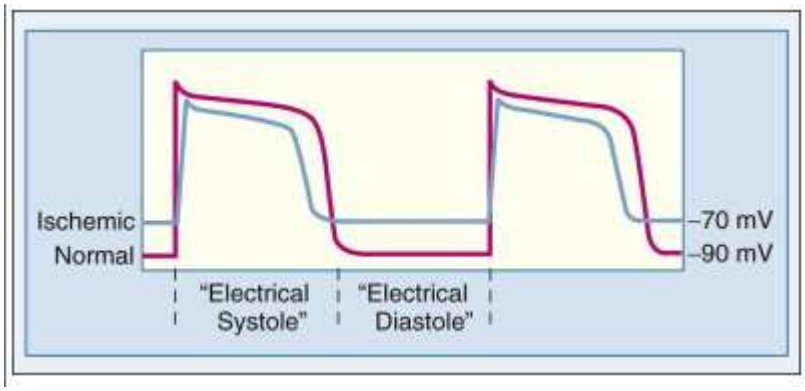
† The exploring electrodes for leads V_5 to V_9 are placed at the same horizontal plane as the electrode for V_4.

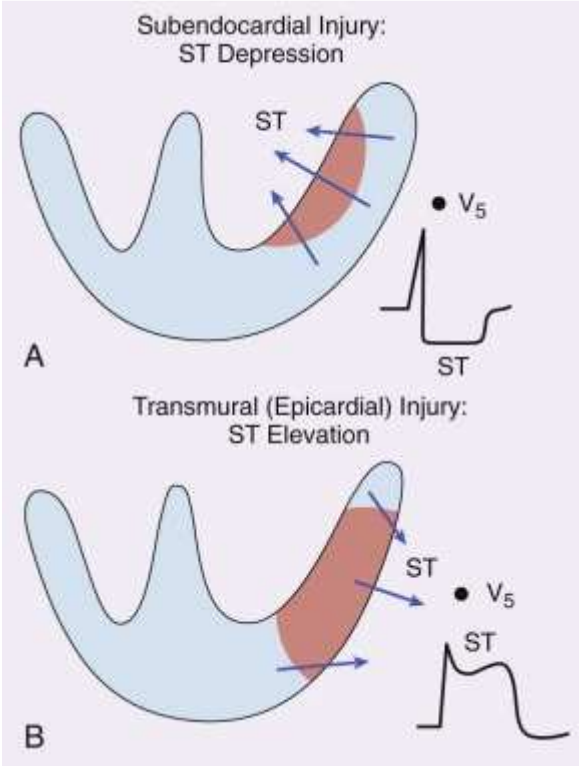
ECG IN MI:

Important diagnostic tool for

1. Duration
2. Extent
3. Topography
4. Any other abnormalities.

MECHANISM OF ST ELEVATION:[38]

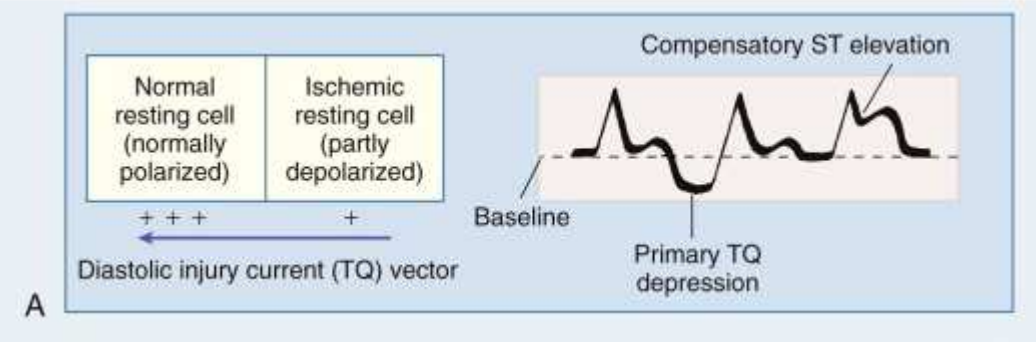




DIASTOLIC CURRENT OF INJURY HYPOTHESIS:

Resting mebrane potential of ischemic cells lowered and in a depolarized state where as non ischemic cells remain in repolarized state. During diastole current flows from depolarized to repolarized segments and hence directed away from negative zone producing TQ segment depression.

As the ECG recorders use AC coupled amplifiers there will be electronic compensation for TQ segment depression as ST segment elevation an apparent shift.



SYSTOLIC CURRENT HYPOTHESIS:

1.Early pathologic repolarization

2.Decreased amplitude of action potential

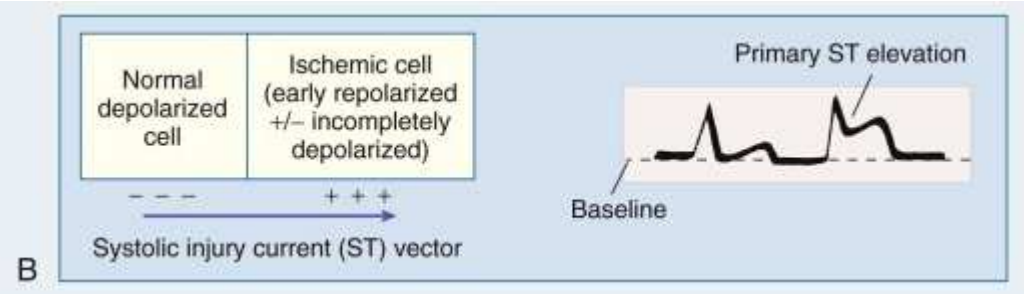
3.Action potential upstroke velocity is decreased.

↓

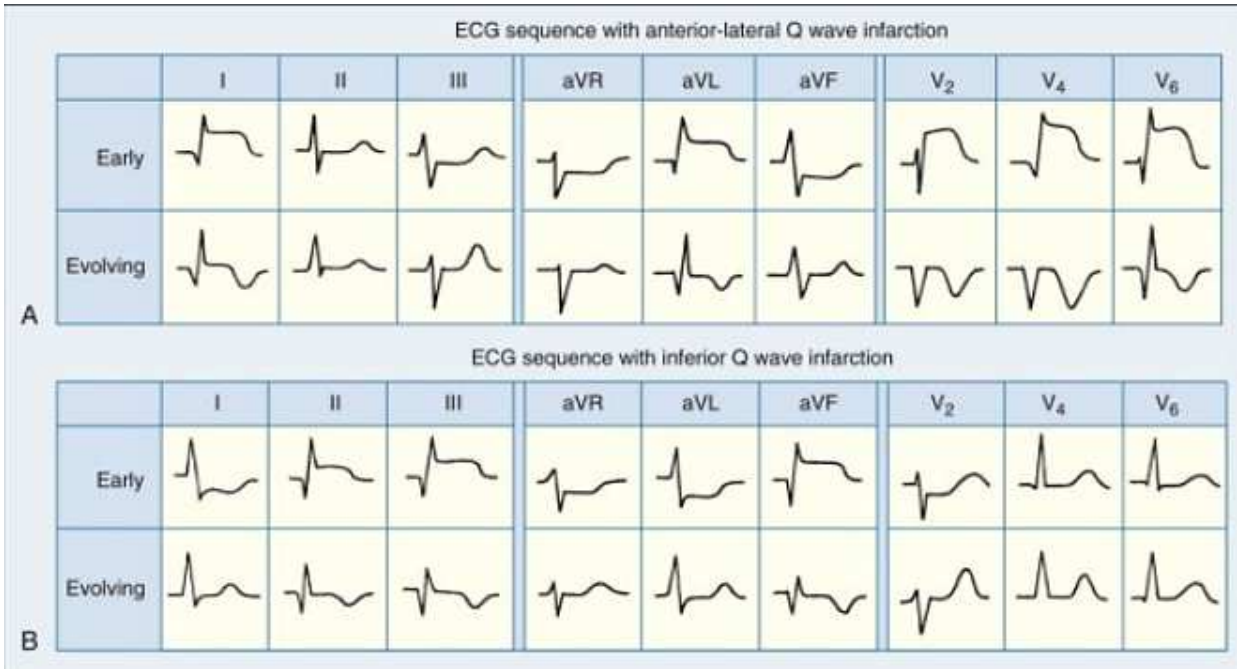
Establishment of voltage gradient between normal and ischemic zone

↓

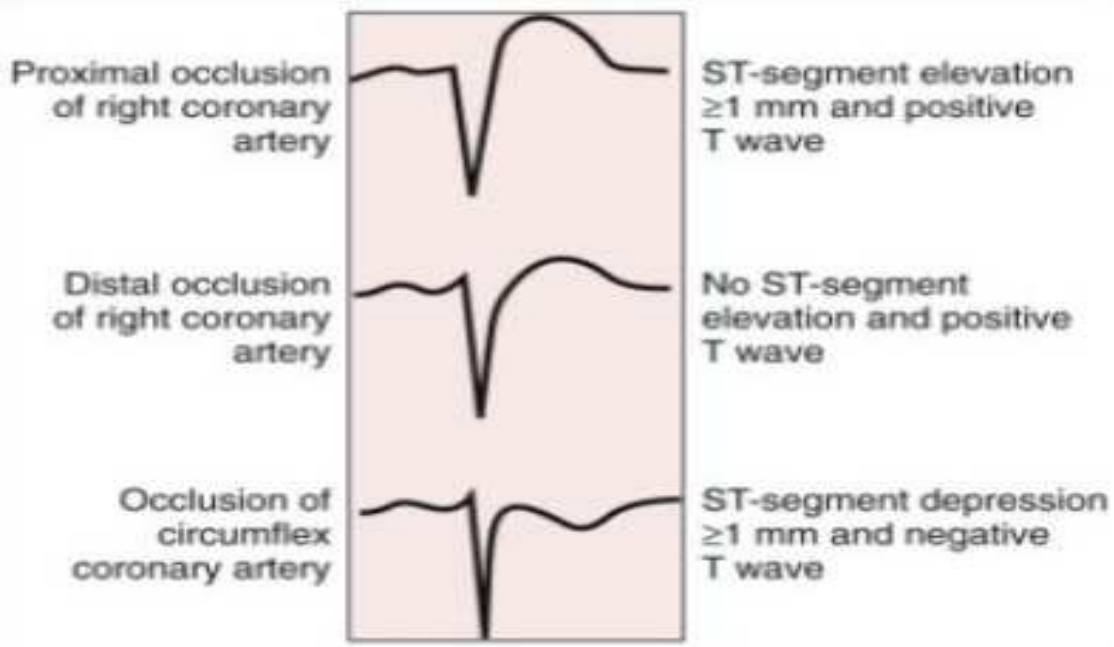
ST SEGMENT ELEVATION



TOPOGRAPHICAL LOCALISATION:[39]



LOCALISATION OF INFERIOR WALL MI:[40]



ECHOCARDIOGRAPHY:[41]

Uses:

To diagnose or exclude MI in case of non diagnostic ECG

Estimation of myocardium at risk and infarct size

Identification of complications

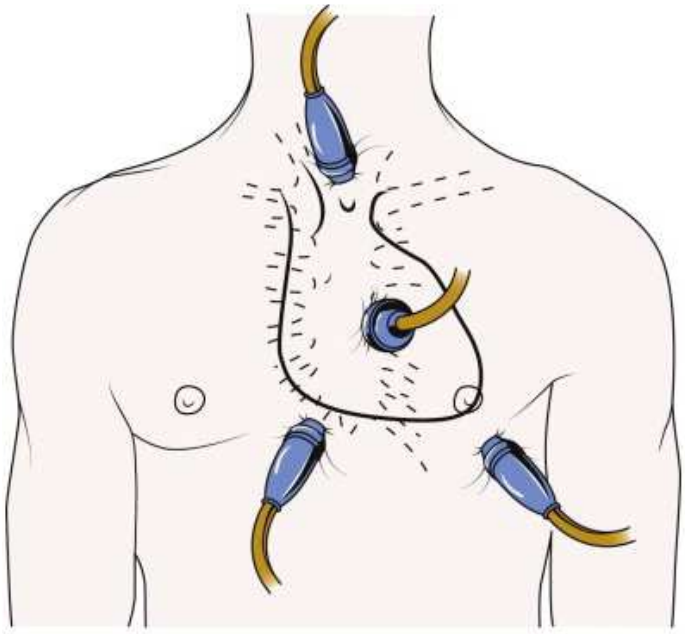
Risk stratification

Assesment of reperfused segments

Principle:[42]

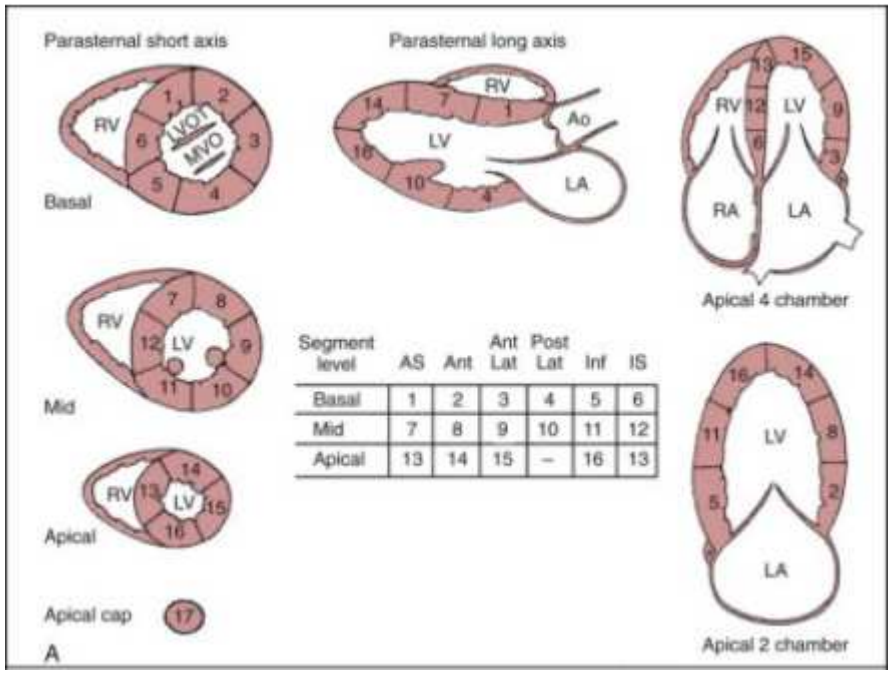
Lines or shapes produced by reflection of ultrasound beams by cardiovascular structures.

Can be taken in one, two,three dimensional mode.



ASE RECOMMENDATIONS:[43]

17 Segment model has been used for regional wall motion analysis.



Segment level	AS	Ant	Ant Lat	Post Lat	Inf	IS
Basal	1	2	3	4	5	6
Mid	7	8	9	10	11	12
Apical	13	14	15	–	16	13

GRADING OF CONTRACTILE FUNCTION:[44]

- GRADE1: Normal >40% thickening with systole
- GRADE2: Hypokinesis(10 to 40%thickening)
- GRADE3:Akinesis <10% thickening
- GRADE4:Dyskinesis
- GRADE5:Aneurysm

WALL MOTION SCORE INDEX[45] =

SUM OF WALL MOTION SCORES /SEGMENTS VISUALISED

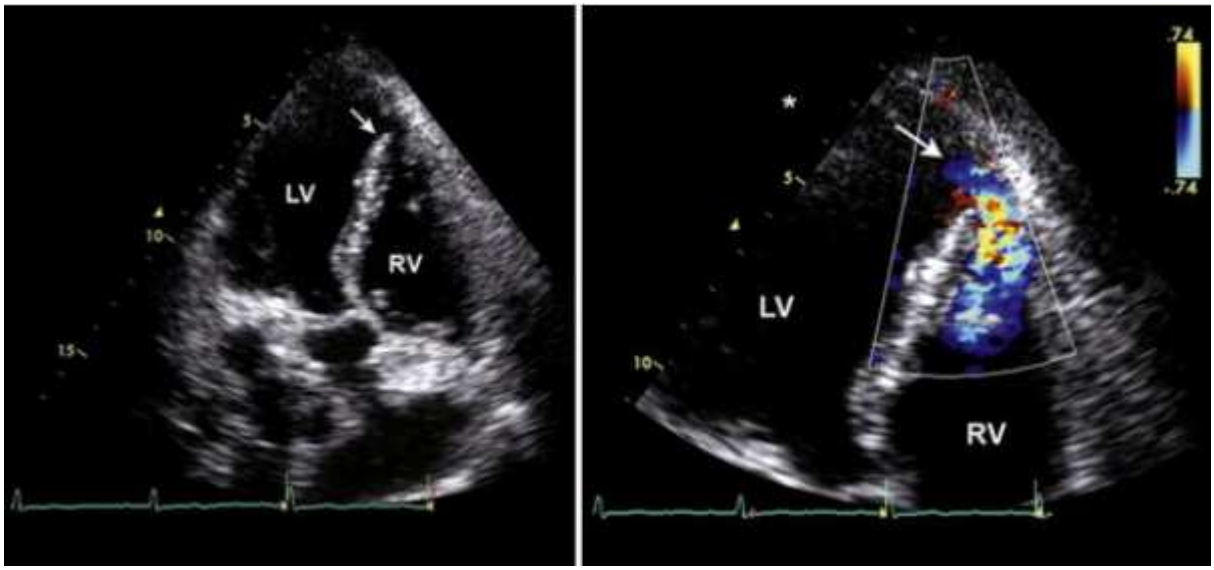
DETECTION OF MECHANICAL COMPLICATIONS:

VENTRICULAR SEPTAL RUPTURE:[46]

Almost always in thinned dyskinetic myocardium

Inferoseptal rupture signifies right ventricular involvement carries a poor prognosis.

Identified by right to left shunt.



ACUTE MR:[47]

Causes:

1.Papillary muscle dysfunction

2.Global or regional LV remodeling

3.Papillary muscle rupture - Posteromedial (supplied by single coronary artery)

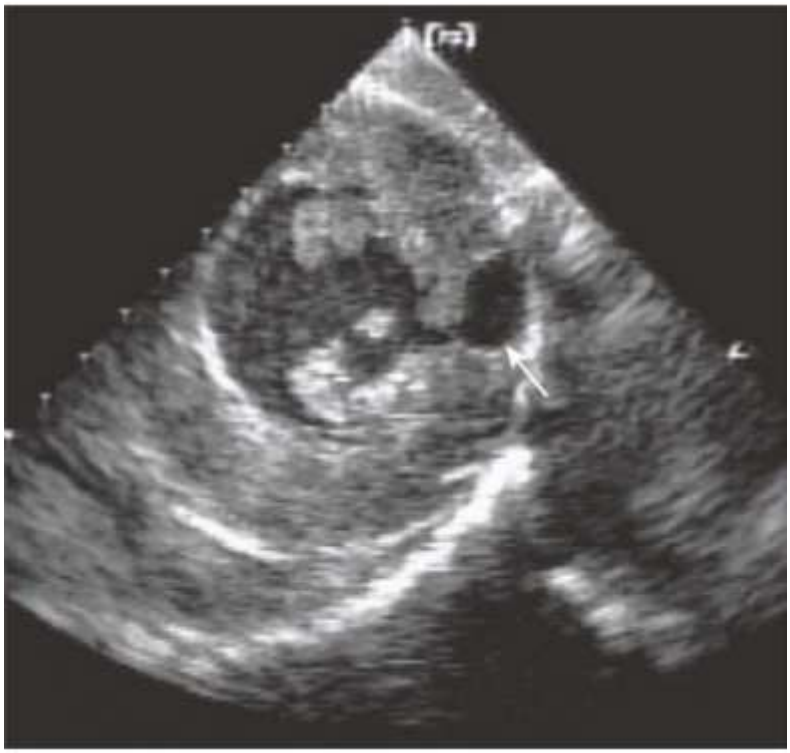
4.Systolic anterior motion of mitral valve

LEFT VENTRICULAR ANEURYSM:[48]

Most common site—Apex >Inferobasal area

Contains all layers of myocardium

Systolic thinning and bulging is common.



THROMBUS:

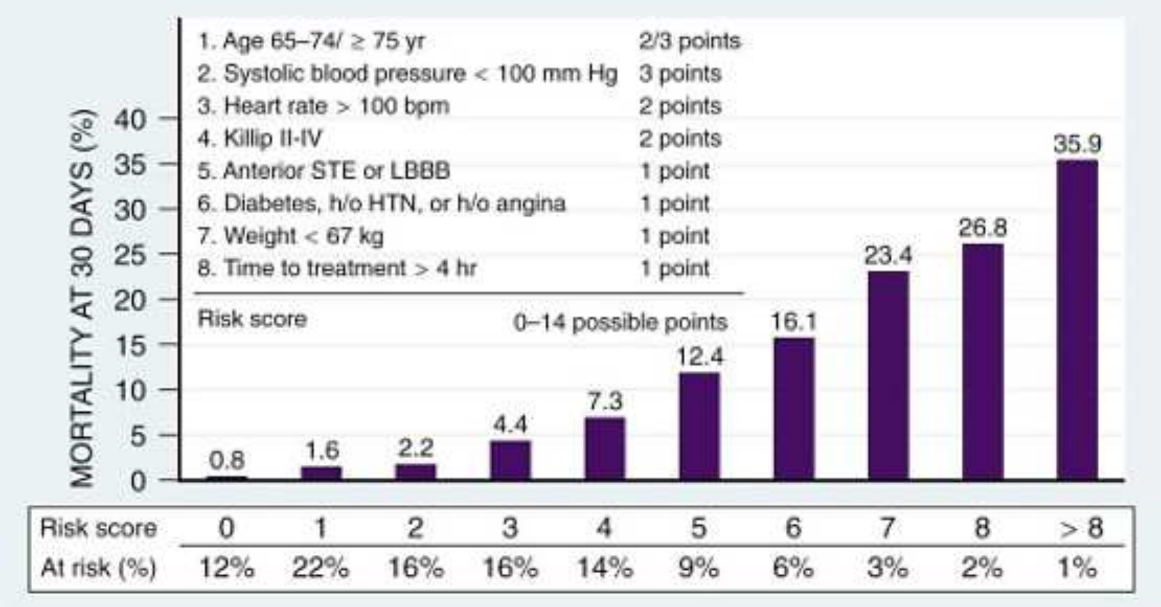
Non homogenous density

Underlies akinetic or dyskinetic wall

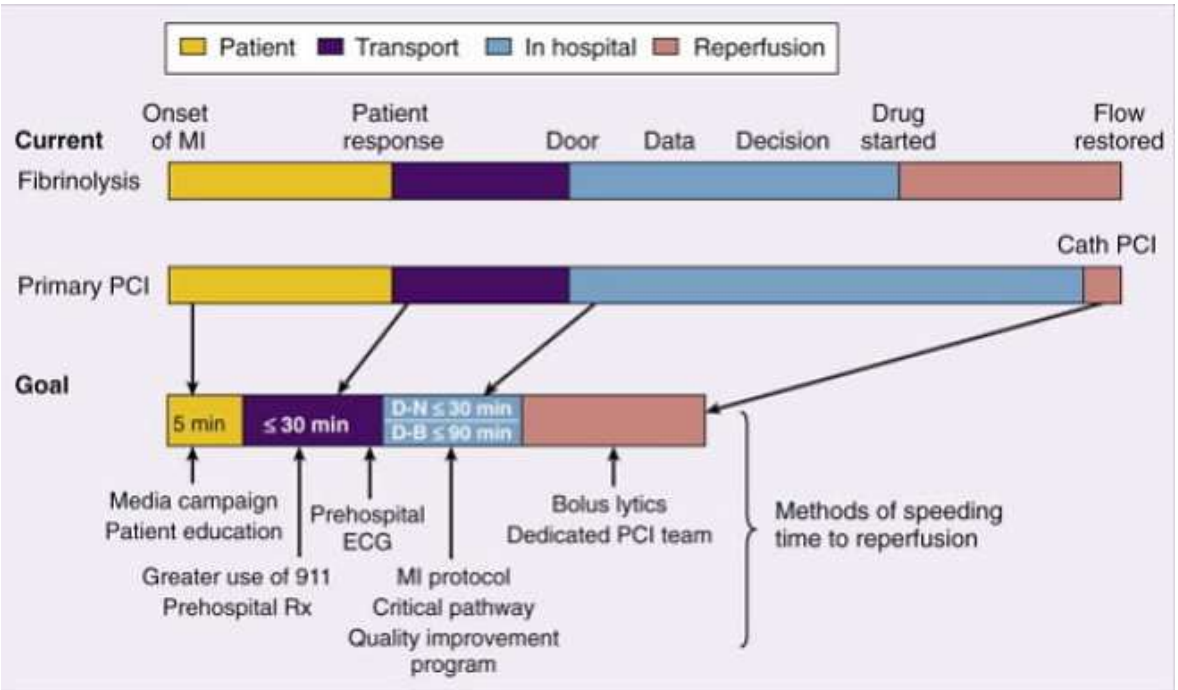
Margins are distinct

Contrast echo needed for confirmation

MORTALITY PREDICTING SCORE (TIMI)[51]



TREATMENT:[49]



ANALGESICS:

Morphine

- Drug of choice
- 4-8mg IV followed by 2-8mg every 15 minutes
- Mechanism of action – peripheral arterial and venous dilatation
- Side effects – hypotension, respiratory depression, vomiting

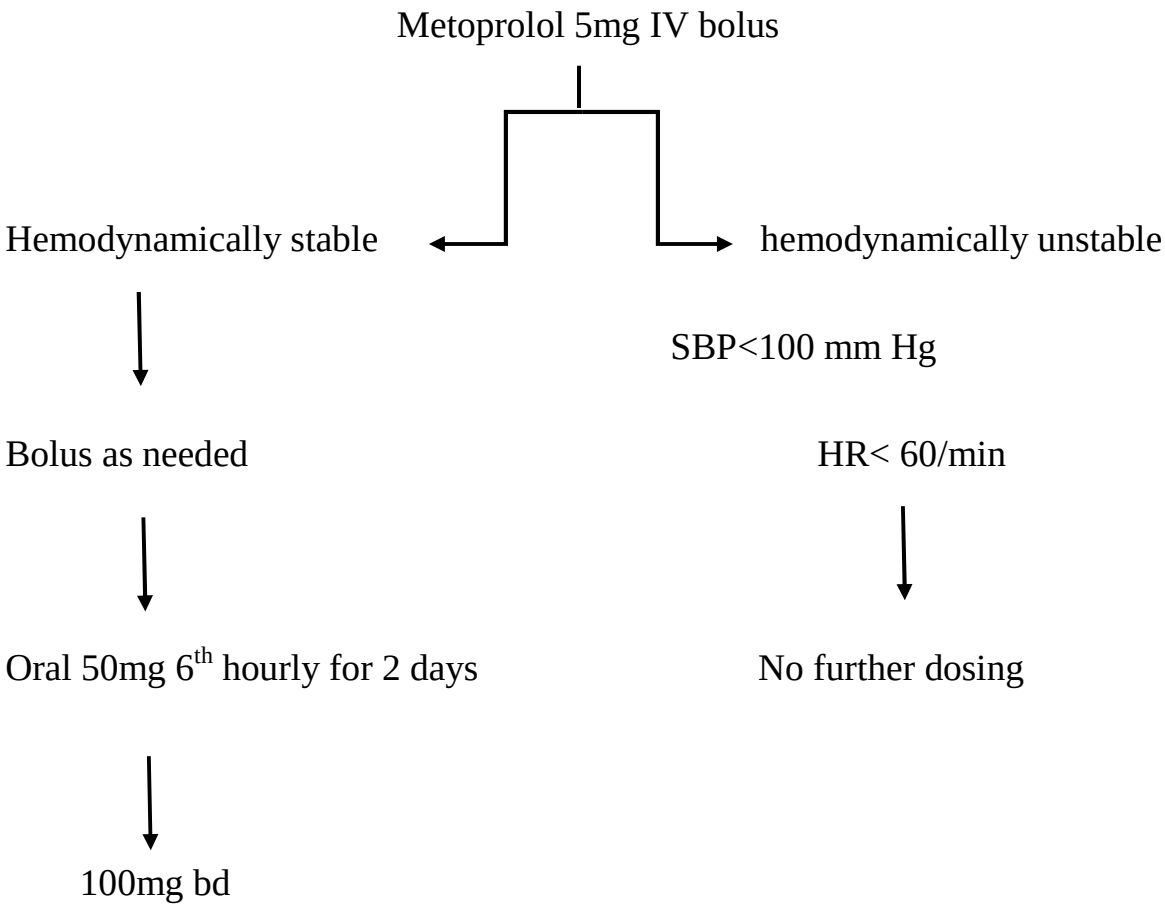
ASPIRIN:

- Loading dose – 160-325mg
- Chewing increases buccal absorption rather than swallowing

NITRATES:

- Mechanism of action – coronary vasodilatation and increased venous capacitance
- Contraindication – right ventricular MI, Systolic BP < 90mmHg and bradycardia

BETA-BLOCKERS:[50]



Contraindication:

- Rales more than 10cm from diaphragm,
- Systolic BP < 90 mm hg
- Heart rate < 60/min
- First degree AV block

OXYGEN:

Only useful if SaO2 < 90%

Mode of administration: 2-4 lit/min 100% O2 by oxygen mask or nasal prongs for 6-12 hours

Oxygen

↓

Increases Systemic vascular resistance & arterial pressure

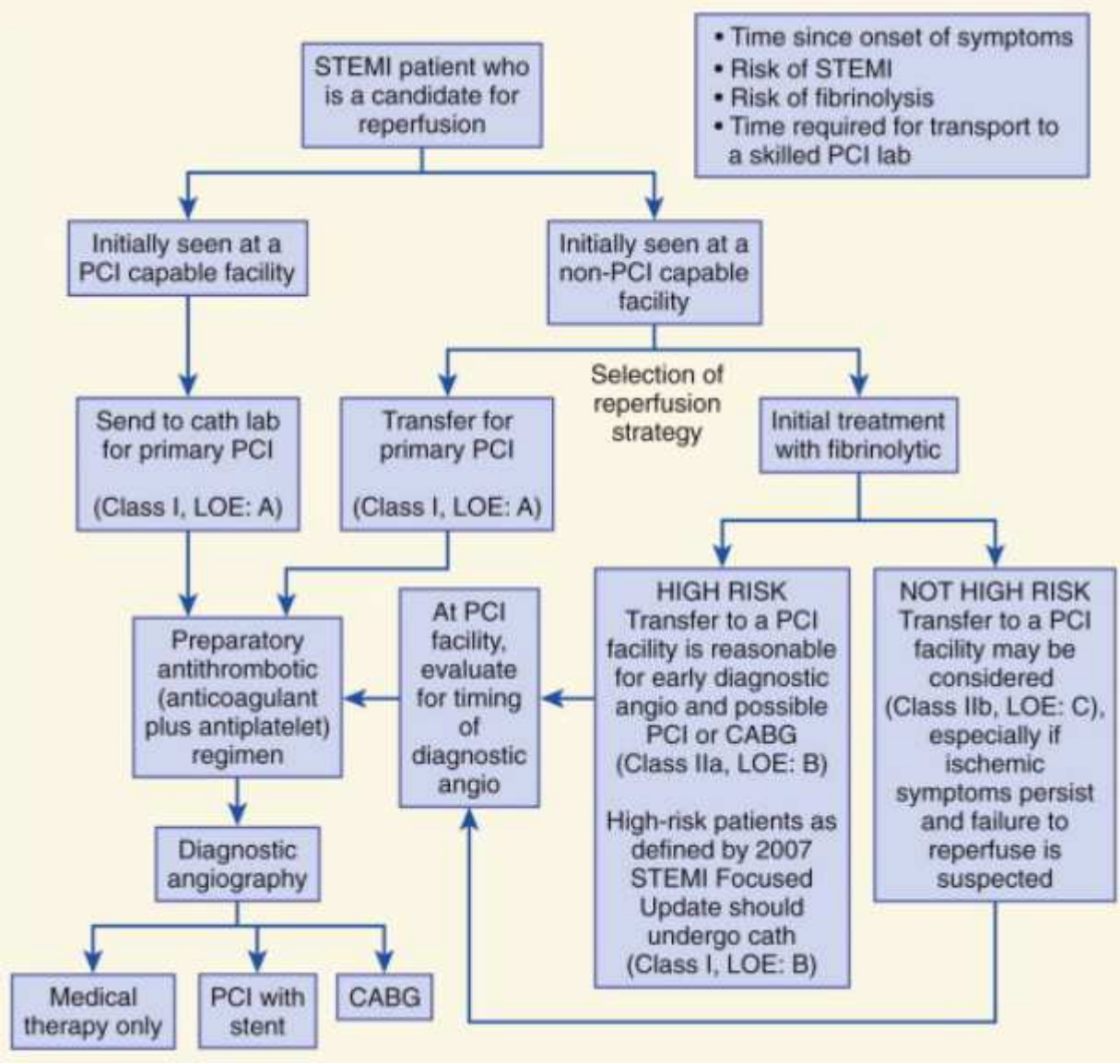
↓

Decreases cardiac output

FIBRINOLYTIC THERAPY:[53]

PARAMETER	STREPTOKINASE	ALTEPLASE	RETEPLASE	TNK t-PA
Dose	1.5 MU in 30-60 min	Up to 100 mg in 90 min (based on weight)	10 U ? 2 (30 min apart) each over 2 min	30-50 mg based on weight
Bolus administration	No	No	Yes	Yes
Antigenic				
Allergic reactions (hypotension most common)	Yes	No	No	No
Systemic fibrinogen depletion	Marked	Mild	Moderate	Minimal
90-min patency rates (%)	≈50	≈75	≈75	≈75[†]
TIMI grade 3 flow (%)	32	54	60	63

APPROACH TO STEMI PATIENT



FIBRINOLYTIC THERAPY:

Absolute Contraindications

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arteriovenous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 mo *except* acute ischemic stroke within 3 hr
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head or facial trauma within 3 mo

Relative Contraindications

- History of chronic severe poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP >180 mm Hg or DBP >110 mm Hg) [†]
- History of prior ischemic stroke > 3 mo, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (>10 min) CPR or major surgery (<3 wk)
- Recent (within 2-4 wk) internal bleeding
- Noncompressible vascular punctures
- For streptokinase, anistreplase: Prior exposure (>5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

ANTICOAGULANTS:[54]

UNFRACTIONATED HEPARIN:

- Used for atleast 48 hours
- Loading dose 60U/kg bolus, followed 12U/kg/hr infusion for 48 hours
- Target aPTT – 1.5 to 2 times the control
- Adverse effects
    - bleeding,
    - heparin induced thrombocytopenia (HIT) – common in females, elderly age

LOW MOLECULAR WEIGHT HEPARIN:[54]

Enoxaparin:

- Age <75 → 30mg IV bolus followed by 1mg/kg bd subcutaneously
- Age >75 → No IV bolus, followed by 0.75mg/kg bd subcutaneously
- Creatinine clearance <30ml/min - 30mg IV bolus, followed by 1mg/kg OD subcutaneously
- Advantages – reocclusion, reinfarction, recurrence rates decreased, high bioavailability, decreased incidence of HIT

Hirudin &Bivalirudin:[55]

- Recurrence of MI reduced by 25-35%
- No mortality reduction
- Adverse effect – stent thrombosis

FACTOR 'X' A ANTAGONISTS:

Fondaparinux:[56]

- 2.5mg subcutaneous – not superior to unfractionated heparin
- Catheter thrombosis when used alone, without another antithrombin agent

ANTIPLATELETS:[57]

Thrombi rich platelets are resistant to fibrinolysis. Hence single antiplatelet agent is not efficient.

Although aspirin inhibits cyclooxygenase pathway, platelet activation continues to occur by thromboxane A2 independent pathways.

P2Y12 inhibitors can be used to prevent platelet aggregation

Clopidogrel – loading dose 300-600mg, maintenance dose 75mg/day

Ticlopidine – loading dose 500mg, maintenance dose 250mg BD

Prasugrel – reduces stent thrombosis 42% compared with clopidogrel

Reversible P2Y12 inhibitor – ticagrelor loading dose 180mg, maintenance dose 90mg BD

ATHEROSCLEROSIS:[58]

It's a chronic inflammatory process

Common sites:

1. CORNARY ARTERIES
2. BIFURCATION OF CAROTID ARTERIES
3. ABDOMINAL AORTA
4. PROFUNDA FEMORIS

RESPONSE TO INJURY HYPOTHESIS:

Endothelial dysfunction is the initiating event that predisposes to atherosclerosis.

ENDOTHELIAL DYSFUNCTION:[60]

Encompasses derangement in any of the following components

1.Vascular tone

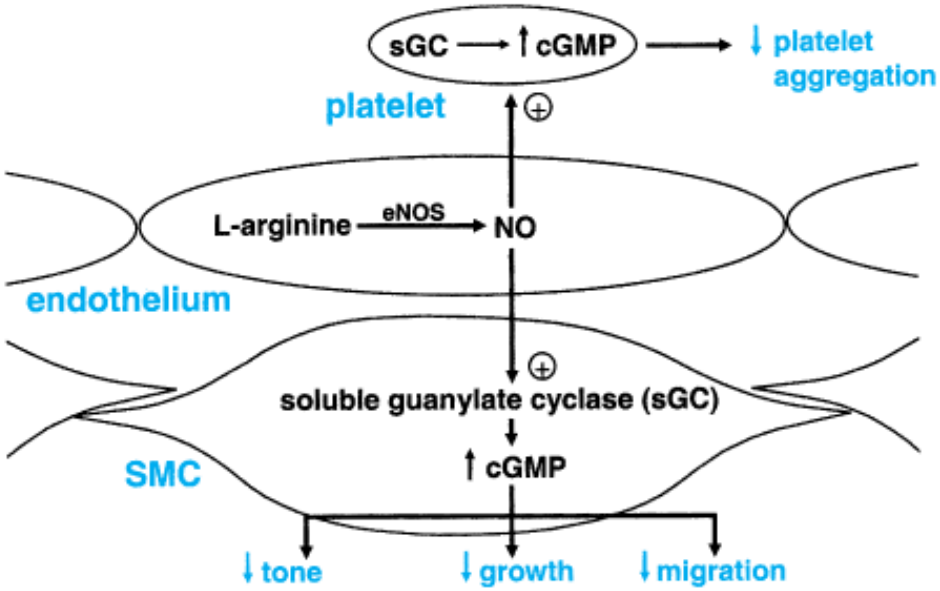
2. Inflammation

3.Growth

4.Preservation of fluidity of blood

VASCULAR TONE:[60]

Normal vascular tone is maintained by nitric oxide



Mechanism of vascular tone derangement

Modified LDL

Nitric oxide synthesis derangements

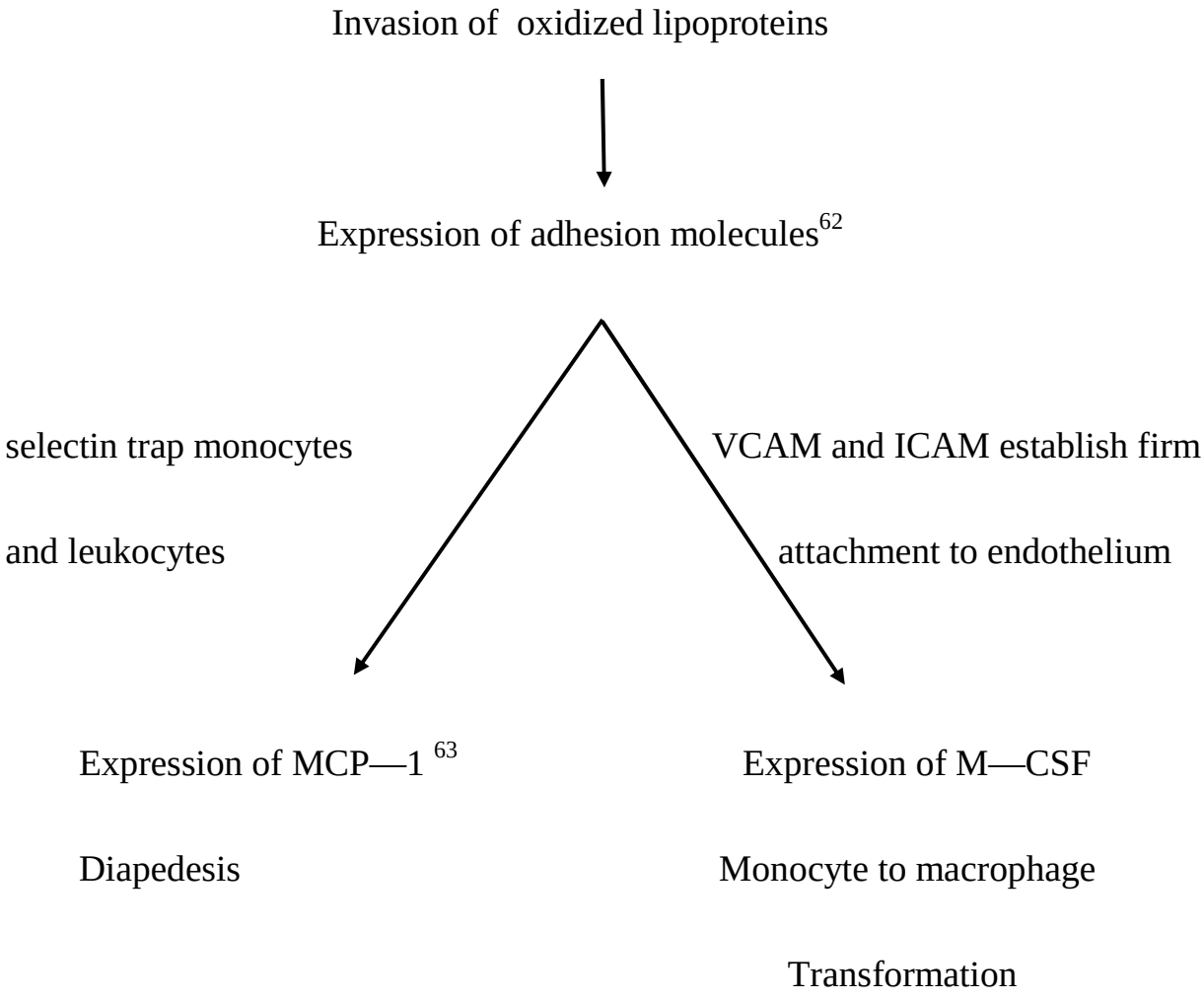
Decreased NO production

High cholesterol

Oxygen free radical

NO inactivation

ENDOTHELIAL INFLAMMATION[61]



ABNORMAL CONTROL OF VASCULAR GROWTH:[64]

Normal endothelium inhibits smooth muscle proliferation

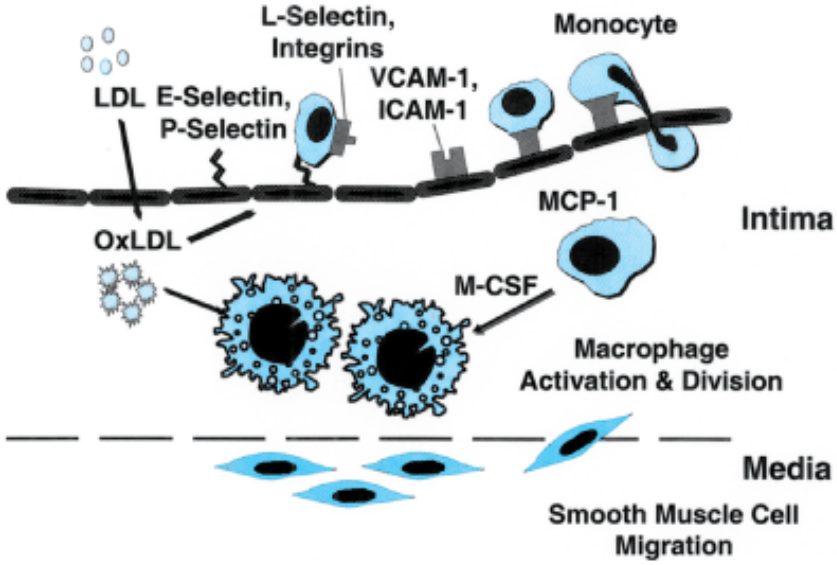
Correct pathogenetic mechanism is unknown

Growth factors like MCP—1 and M-CSF may be responsible for proliferation of smooth muscle

Inflammation induced NO bioactivity decrease also involved in smooth muscle proliferation.

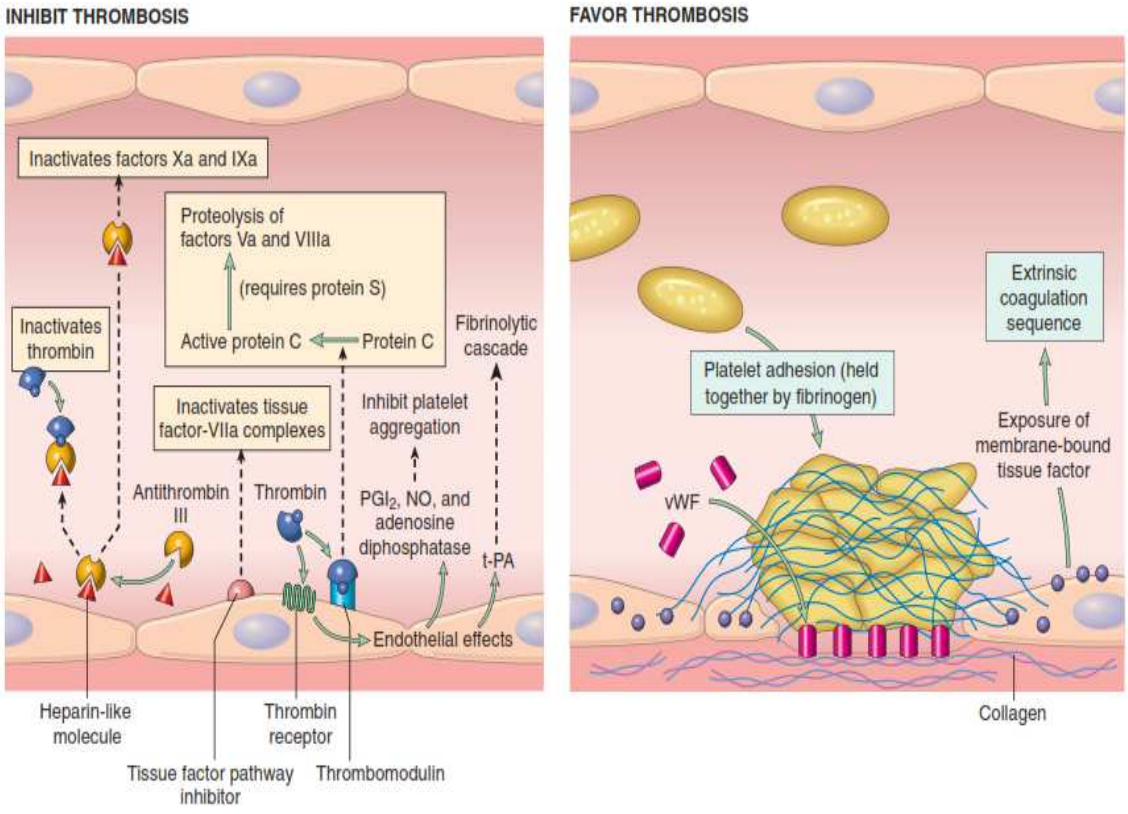
Stability of plaque is given by smooth muscle proliferation

Shoulder region apoptosis of smooth muscle leads to unstable vulnerable plaque.

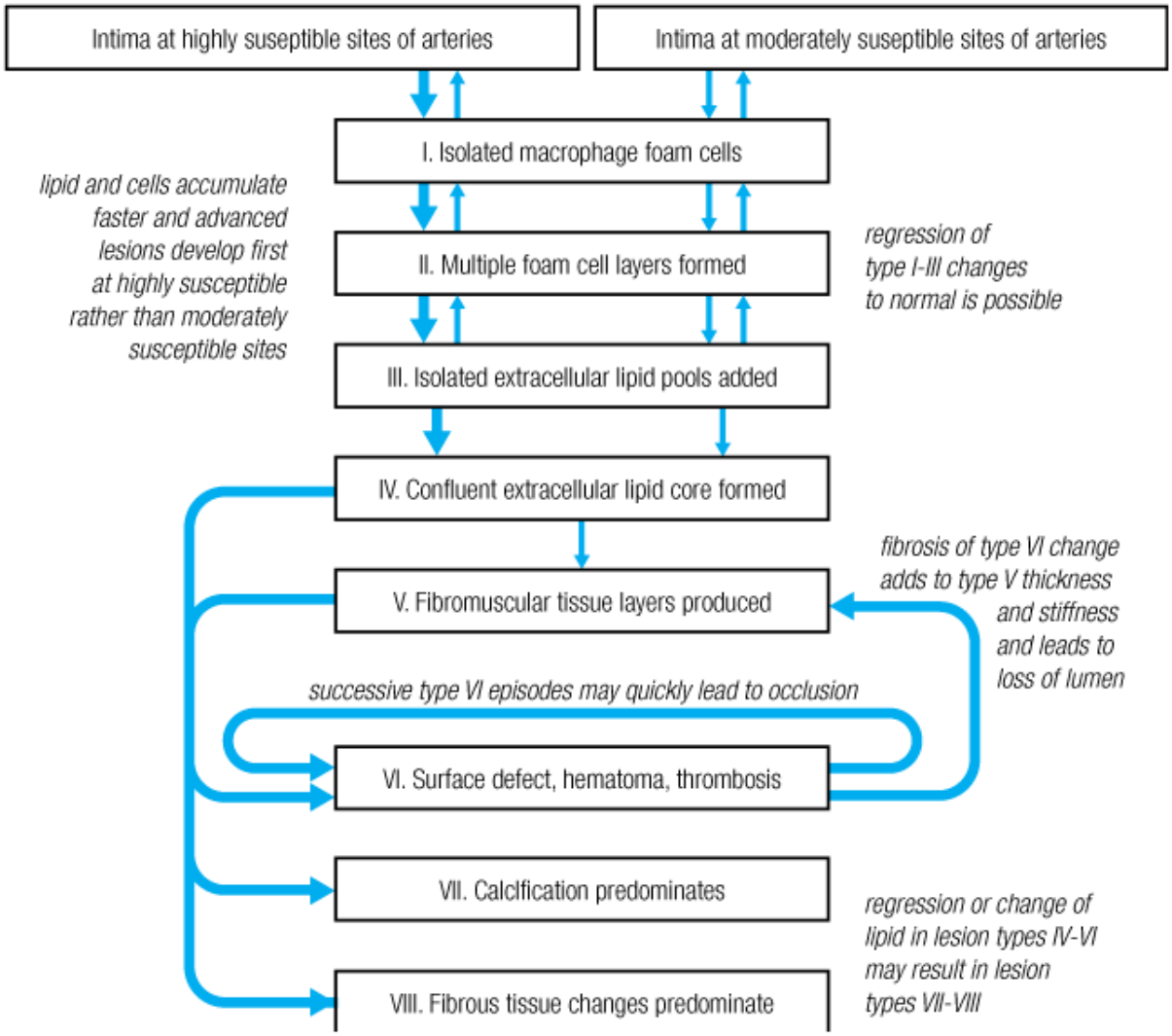


ABNORMAL CONTROL OF BLOOD FLUIDITY:[66]

Imbalance between factors inhibiting and favouring thrombosis leads to prothrombotic state and thrombosis


INHIBIT THROMBOSIS
Inactivates factors Xa and IXa
Proteolysis of factors Va and VIIIa
(requires protein S)
Active protein C
Protein C
Inactivates thrombin
Fibrinolytic cascade
Inactivates tissue factor-VIIa complexes
Inhibit platelet aggregation
Antithrombin III
Thrombin
PGI2, NO, and adenosine diphosphatase
t-PA
Endothelial effects
Heparin-like molecule
Thrombin receptor
Tissue factor pathway inhibitor
Thrombomodulin
FAVOR THROMBOSIS
Extrinsic coagulation sequence
Platelet adhesion (held together by fibrinogen)
Exposure of membrane-bound tissue factor
vWF
Collagen

AHA ATHEROSCLEROTIC PLAQUE CLASSIFICATION[67]

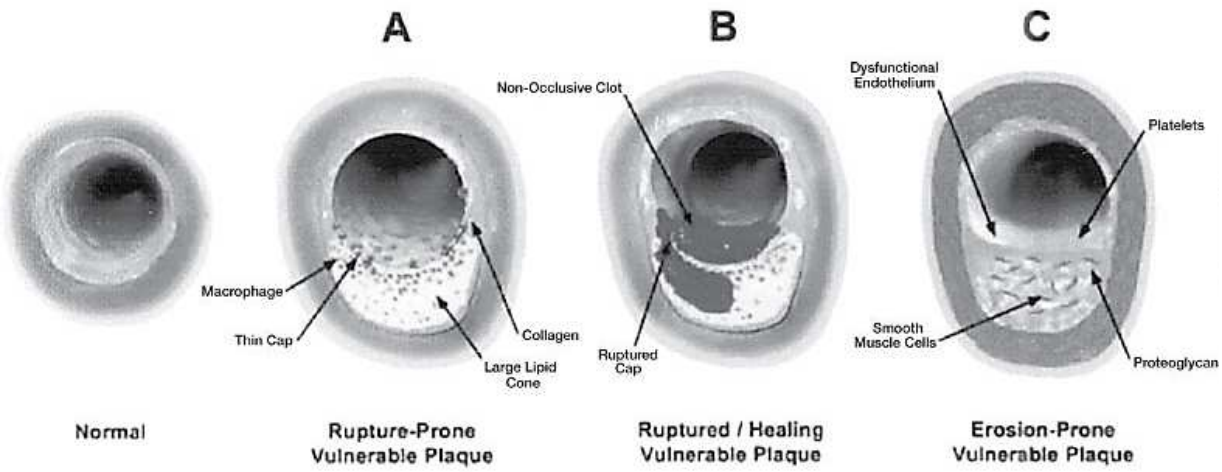


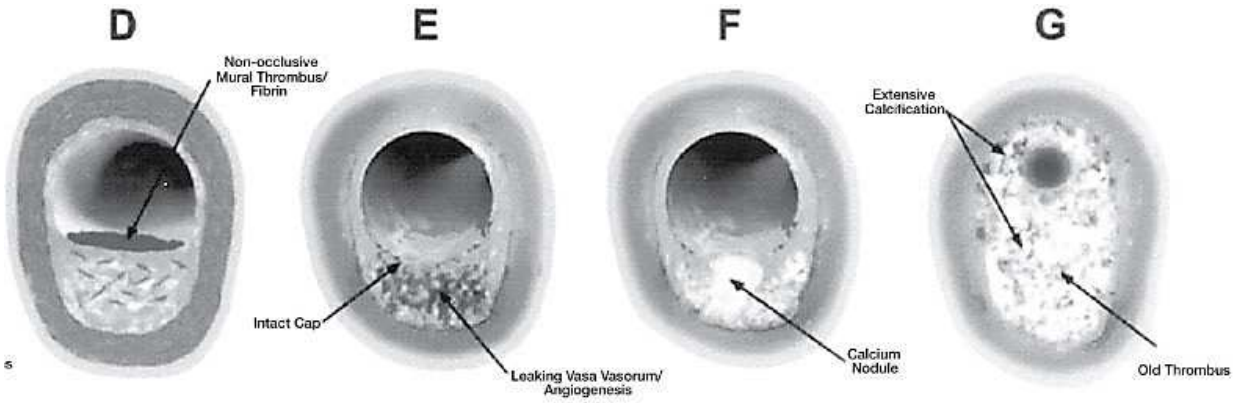
VULNERABLE PATIENT:

Combination of vulnerable plaque, blood and myocardium

Vulnerable plaque:[69]

Major criteria
Active inflammation (monocyte/macrophage and sometimes T cell infiltration)
Thin cap with large lipid core
Endothelial denudation with superficial platelet aggregation
Fissured plaque
Stenosis >90%
Minor criteria
Superficial calcified nodule
Glistening yellow
Intraplaque hemorrhage
Endothelial dysfunction
Outward (positive) remodeling





Markers of blood vulnerability[71]

1. Markers of blood hypercoagulability
Decreased anticoagulation factors (e.g., proteins C and S, antithrombin)
Prothrombotic gene polymorphisms (e.g., factor V Leiden, G20210A prothrombin mutation)
Increased coagulation factors (e.g., fibrinogen, factors VII and VIII, von Willebrand factor)
2. Increased platelet activation
(e.g., gene polymorphisms of platelet integrin $\alpha_{IIb}\beta_3$, integrin $\alpha_2\beta_1$, GpIb-IX)
3. Decreased endogenous fibrinolysis activity
(e.g., reduced t-PA, increased PAI-1, certain PAI-1 polymorphisms)
4. Other thrombogenic factors
(e.g., anticardiolipin antibodies, thrombocytosis, sickle cell disease, polycythemia, diabetes mellitus, hyperhomocysteinemia, hypercholesterolemia)
5. Increased viscosity
6. Transient hypercoagulability
(e.g., smoking, dehydration, infection, adrenergic surge, cocaine, estrogens, postprandial.)

THROMBOPOIESIS:[72]

1×10^{11} produced every day

Single megakaryocyte produces 1000—3000 platelets

Half life of platelet—10 days

Platelet count is inversely proportional to thrombopoietin levels

Origin of platelets from megakaryocytes

The megakaryocyte is a large hematopoietic cell, the cytoplasm of which fragments to form circulating blood platelets. The histogenesis of platelets from megakaryocytes was first described by James Wright in 1910. The megakaryocytes are sessile polyploid cells which in turn descend from diploid pluripotent hematopoietic stem cells of marrow.[73] The megakaryocytes are imprisoned within the sub endothelial layer of marrow sinuses by their very girth and volume (average 5000 femto litres, Zhang YJ, 1991). In these marrow niches, mononuclear progenitors undergo diploid doublings by the unique process of endomitosis. Subsequently the polyploidy megakaryocytes accumulate a bulky compartmentalized cytoplasmic mass with large volumes that at end stage maturation disintegrates abruptly to yield between 1000 and 8000 platelets having a volume of 7-9 femto litres each (Martin et al, 1982; Stenberg and Levin,

1989;Corash, 1989). Megakaryocytes are suicidal micro organs whose mission is to proliferate and then fragment their cytoplasm on demand to maintain blood platelets at relatively steady levels of about 1,50,000-3,50,000/mm3.

Maintenance of platelet counts within this range represents a surplus of over 10 times that is necessary to ensure routine haemostasis but provides a precautionary reserve for times of excess platelet loss or consumption

Mechanism of formation:

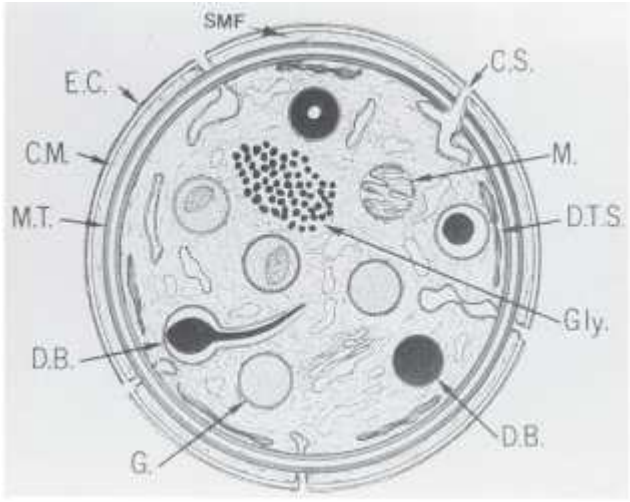
Megakaryocyte

Fragmentation of membranes

Proplatelets

ELECTRON MICROSCOPY:[75]

Glycocalyx composed of glycoproteins, glycolipids, and mucopolysaacharides. Platelets contains sialic acid which contributes to negative charge and prevents adhering from one another and also to the endothelium which is also negatively charged.



Plasmamembrane:

Trilaminar structure

Outer platelet membrane—Contains receptors for interaction

Phospholipids --57% of total content

Inner membrane

Phosphatidyl serine—negatively charged

ORGANELLES:

Peroxisomes:[76]

Plasmalogen synthesis

Platelet activating factor synthesis

Mitochondria:[77]

4—7 in number

Hyperglycemia

Superoxide

Platelet aggregation

Lysosomes:

Contains acid hydrolases, β glucuronidases,cathepsin,aryl sulfatase, collagenase

LAMP 1,2,3—markers of platelet release reaction

Dense bodies:[78]

20—30 nm in diameter

Highly osmophilic

ATP:ADP—2:3

Contains serotonin in high concentration

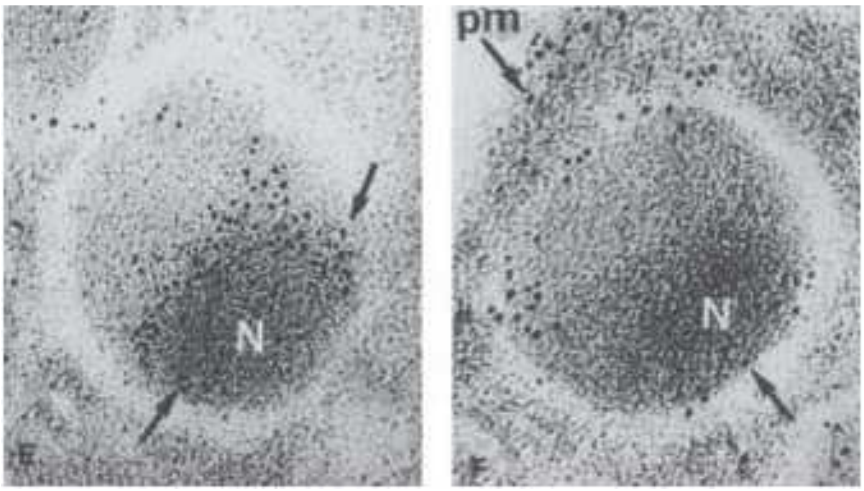
Also contains calcium, magnesium

Alpha granules:[79]

Most abundant granules

50—80 per platelet

200 nm in diameter



Nucleoid:

Eccentric accentuated electron density containing β-thromboglobulin, platelet factor –4 proteoglycans.

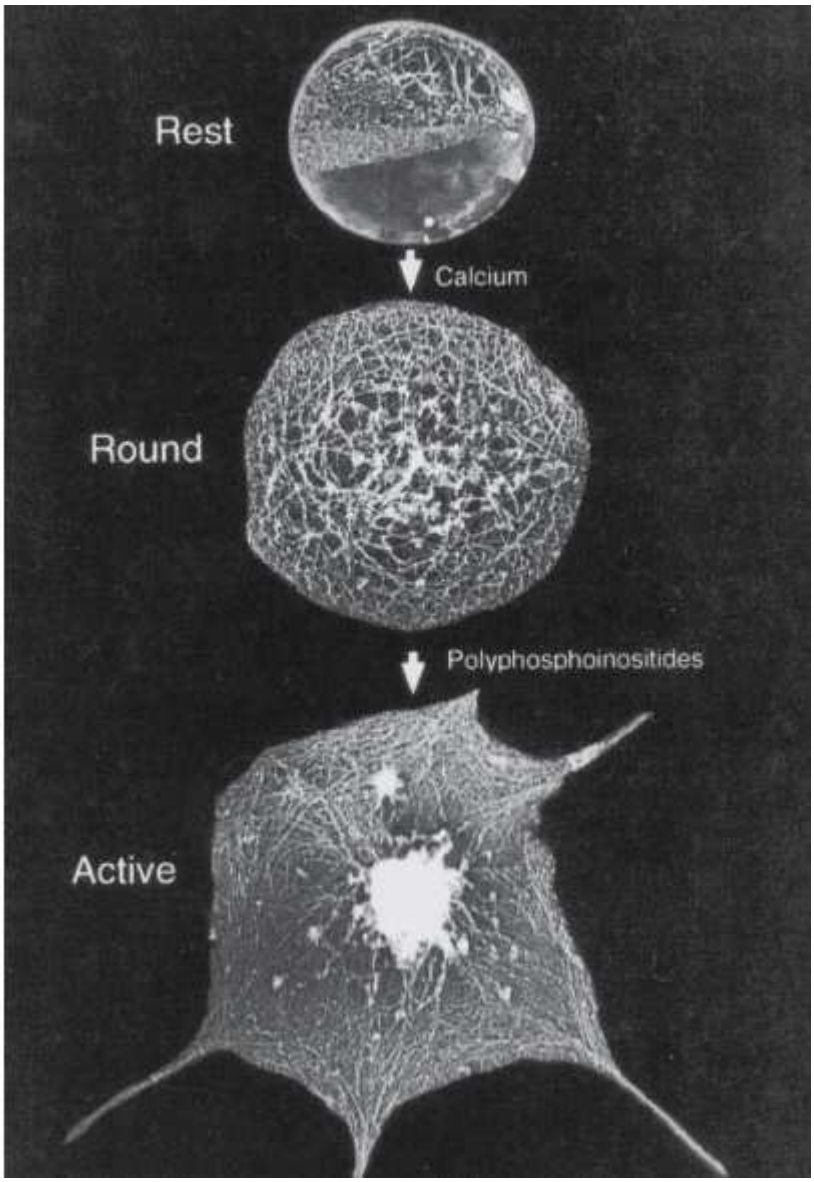
Electron lucent areas rich in Vwf, multimerin, Factor V

Cytoskeleton[80]

General structure: The platelet cytoskeleton contains 30 to 50% of total platelet protein and is made up of three major structural components: an actin microfilament network present throughout the cytoplasm, a microtubule coil localized at the platelet periphery and a membrane skeleton comprising a

network of short actin filaments that underlies the inner surface of the plasma membrane. Although they are distinct structures, interconnections between these elements are present.

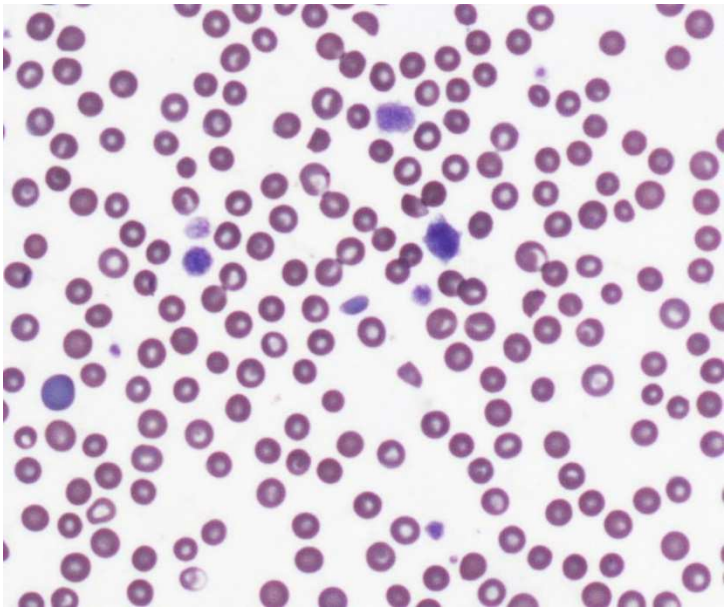
Upon platelet activation, the proportion of filamentous actin rapidly increases to 60-70%. Polymerization of actin monomers at platelet peripheries to form filopodia.



LIGHT MICROSCOPY:[82]

Blood films made from EDTA anticoagulation platelets appear as small round bluish gray particles with purple granules.

Mean diameter—1.5--3µm



PHYSIOLOGY OF THROMBUS FORMATION:[84]

Hemostatic system is maintained in well controlled fashion.

Exposure of normally concealed blood vessel wall by intact endothelial lining leads to dynamic process of platelet adhesion, activation,aggregation.

Atherosclerosis, plaque rupture/erosion

↓

vWF and collagen exposure

↓ GbIb-IX receptor

Adhesion

↓

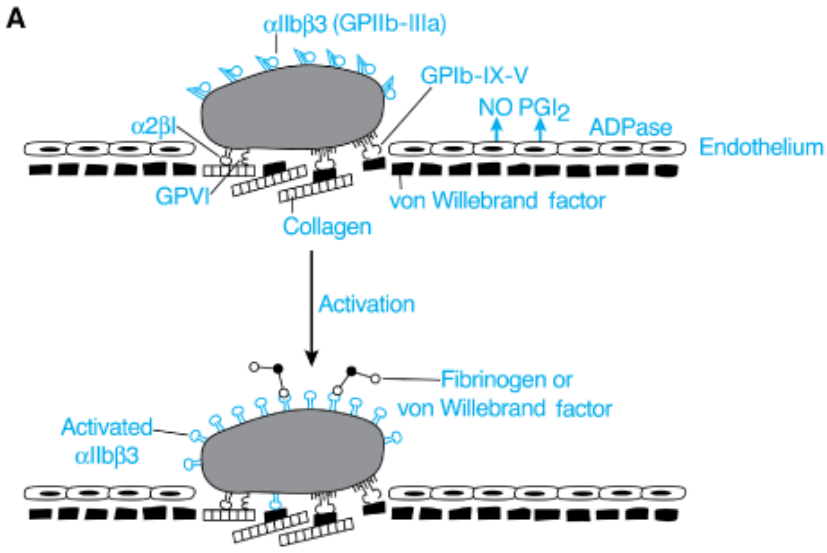
Activation

Confirmational change in œIIβ3

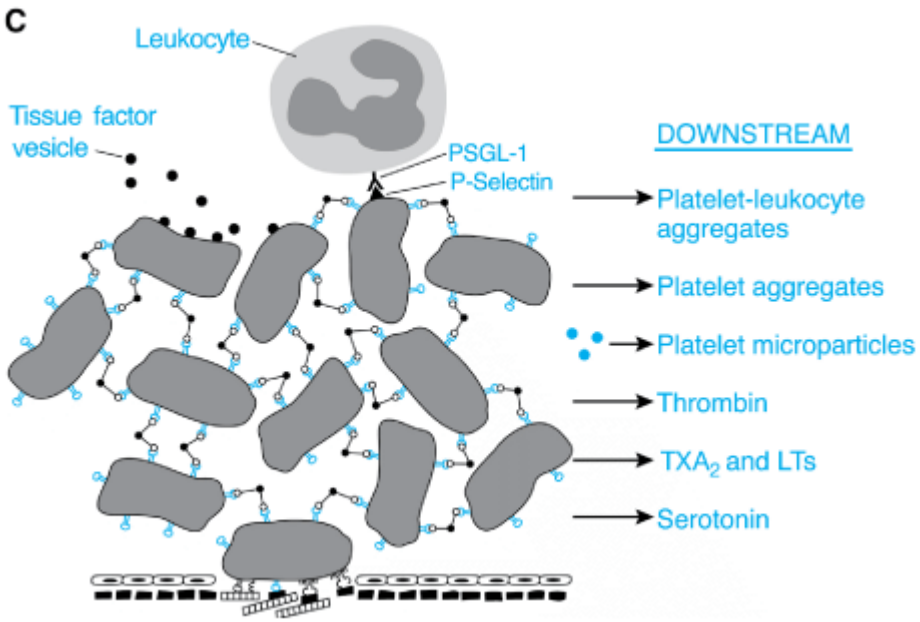
↓

Receptor cross linking

Aggregation



PLATELET –LEUKOCYTE INTERACTION:



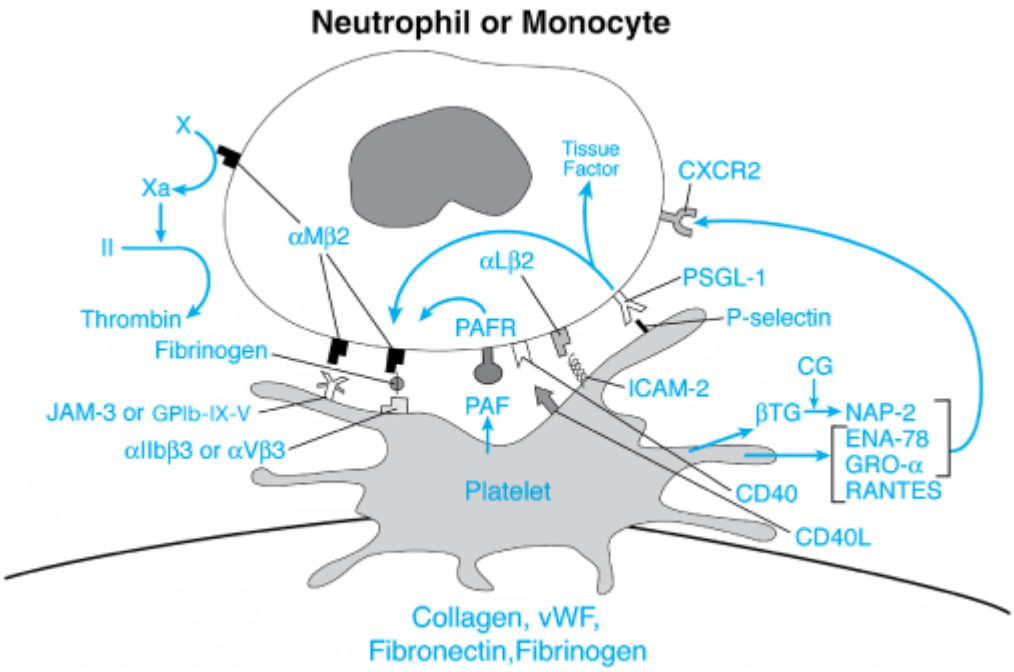
P selectin + PSGL-1[83]

↓

Stimulates monocytes

↓

Release tissue factor



Fibrinogen + αMβ2 in leukocytes (or) αIIβ3 in platelets

↓

Binds and activates factor X

↓

Activates factor II

↓

Thrombin

↓

Fibrinogen → fibrin polymer (crosslinked fibrin clot)

↓

Coronary artery occlusion

CORONARY ARTERIOGRAPHY:

Angiography of coronary arteries is the standard diagnostic procedure to identify atherosclerotic disease induced anatomical changes. First performed in 1959 by SONES.[85]

TECHNIQUE[86]

PREPARATION OF THE PATIENT

Tests to be done

1.Baseline electrocardiogram

2.Complete blood count

3.Coagulation panel

4.Renal function test

5.Electrolytes

Drugs to be stopped:

Warfarin should be stopped before 2days

- INR <2 --Transfemoral approach
- INR<2.5 –Transradial approach

Patients having high risk of thromboembolism like atrial fibrillation, mitral valve disease,previous thromboembolism history can be treated with

low molecular weight heparin subcutaneously or intravenous unfractionated heparin.

PCI undergoing patients should receive Aspirin 162—325mg 2 hours before the procedure.

VASCULAR ACCESS[88]

FEMORAL ARTERY – Most commonly used

Right and left can be used

Site of puncture:

Common femoral artery anterior wall

Centimeters below inguinal ligament –To prevent retroperitoneal hemorrhage.

Proximal to the bifurcation of profunda and superficial femoral artery - to prevent pseudoaneurysm.

Femoral artery and vein cannulation ipsilaterally avoided to prevent arteriovenous fistula.

RADIAL APPROACH:[87]

Preffered due to easy catheter entry and removal.

Allen test –Ulnar artery patency.

2000---5000 U Unfractionated heparin or Bivalirudin –Prevent thrombosis.

Intraarterial verapamil or nitrglycerine –Prevent spasm.

Advantages:

- Low cost.
- Better coronary visualization.
- Less bleeding complications.

CATHETERS

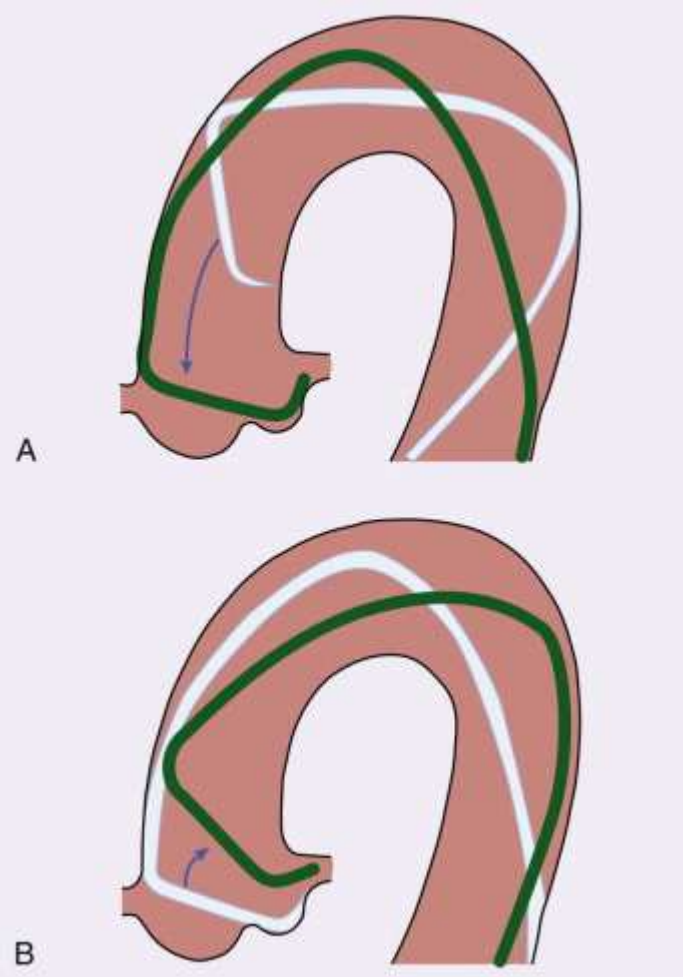
Made from polyurethane or polyethelyne.

Outer diameter size 4F –8F.

5F OR 6F –Commonly used.

JUDKINS OR AMPLATZ CATHETER.[89]

Size determined by bodyhabitus and aortic root size



DRUGS DURING THE PROCEDURE:

ANALGESICS:

Diazepam 2.5—10mg plus Diphenhydramine 25—50mg oral 1 hour before the procedure (or)

Intravenous midazolam 0.5—2mg plus fentanyl25 –50micrograms

ANTICOAGULANTS:

Intravenous unfractionated heparin 2000—5000units intravenously in high risk patients, severe aortic stenosis.

Heparinised saline flush –Prevents microthrombi

Reversal of heparin 100units by 1mg protamine.

Protamine contraindicated in previous catheterization by radial or femoral artery, NPH Insulin use,unstable angina, complex coronary anatomy.

Periprocedural ischemia:

Causes:

1.Tachycardia

2.Hypertension

3.Contrast agents

4.Dynamic platelet aggregation.

5.Coronary vasospasm.

6. Increased vasomotor tone.

TREATMENT:

1.Nitroglycerine 0.3mg sublingually or50 –200 microgram intracoronary or 10—25microgram/min intravenous.

2.Intravenous metoprolol 2.5—5mg or propranolol 1—4mg.

3. Intraaortic balloon counterpulsation.

CONTRAST AGENTS

Principle:

IODINE

↓

X ray absorption

Energy range

High - osmolar ionic agents:

1.Sodium diatrizoate

2. Sodium meglumine diatrizoate

Adverse effects:

Due to hypertonicity.

1.Sinus bradycardia.

2.QT Prolongation.

3.QRS Prolongation.

4.ST Depression.

5. Ventricular fibrillation.

Non ionic low osmolar contrast agents:

1.Ioxaglate.

2.Iohexol.

3.Iopamidol.

4.Ioversol.

5. Iodixanol

Side effects:

1.Hotflush.

2.Vomiting.

3.Hypotension—Anaphylaxis

Contrast induced nephropaty:[90]

Risk factors:

1.Prior renal insufficiency.

2.Diabetes mellitus.

3.Dehydration.

4.Large volume of contrast material.

Prevention:

1.Prior hydration.

2.Limited contrast usage.

Contrast reactions grading:[91]

Grade1:

Vomiting, nausea, vertigo—one episode.

Grade2:

Hives >1 episode of fever, chills, vomiting.

Grade3:

Bronchospasm, laryngospasm, hypotension, loss of consciousness, arrhythmia, pulmonary edema.

Prevention:

1.Prednisone 60mg(night and2 hours before procedure).

2.Diphenhydramine50mg.

3. Cimetidine 300 mg.

ARTERIAL NOMENCLATURE:

SCORING SYSTEMS:[93]

1. **CALIFF SCORING:**

   Coronary segments-6

   Score-0-12

2. **CANDELL-RIERA SCORING**:

   Coronary segments-13

   Score-0-65

3. **GENSINI SCORING:**

   Cornary segments-11

   Score-0-72

CORONARY ARTERY SURGERY STUDY (MODIFIED BY BYPASS ANGIOPLASTY REVASCULARISATION INVESTIGATORS)[92]

29 segments of coronary artery are defined.

NUMBER	MAP LOCATION	NUMBER	MAP LOCATION	NUMBER	MAP LOCATION
Right Coronary Artery (RCA)		Left Main Coronary Artery		Left Circumflex Artery (LCx)	
1	Proximal RCA	11	Left main coronary artery	18	Proximal LCx
2	Mid RCA	Left Anterior Descending (LAD)		19	Distal LCx
3	Distal RCA	12	Proximal LAD	20	First obtuse marginal
4	Right posterior descending branch	13	Mid LAD	21	Second obtuse marginal
5	Right posterior atrioventricular	14	Distal LAD	22	Third obtuse marginal
6	First right posterolateral	15	First diagonal	23	LCx atrioventricular groove
7	Second right posterolateral	16	Second diagonal	24	First left posterolateral
8	Third right posterolateral	17	LAD septal perforators	25	Second left posterolateral
9	Posterior descending septals	29	Third diagonal	26	Third left posterolateral
10	Acute marginal segment	27	Left posterior descending branch		
		28	Ramus intermedius branch		

CORONARY ARTERY DISEASE:

Definition:

- Obstructive coronary artery disease: >50% stenosis
- Non obstructive coronary artery disease
- Subcritical stenosis: <50% stenosis

LESION CHARACTERS:[94]

LESION-SPECIFIC CHARACTERISTICS	
Type A Lesions (high success, >85%; low risk)	
Discrete (<10 mm)	Little or no calcium
Concentric	Less than totally occlusive
Readily accessible	Not ostial in locations
Nonangulated segment, <45 degrees	No major side branch involvement
Smooth contour	Absence of thrombus
Type B Lesions (moderate success, 60%-85%; moderate risk)	
Tubular (10 to 20 mm in length)	Moderate to heavy calcification
Eccentric	Total occlusions <3 months old
Moderate tortuosity of proximal segment	Ostial in location
Moderately angulated segment, ≥45 degrees, <90 degrees	Bifurcation lesion requiring double guidewire
Irregular contour	Some thrombus present
Type C Lesions (low success, <60%; high risk)	
Diffuse (>2 cm in length)	Total occlusion >3 months old
Excessive tortuosity of proximal segment	Inability to protect major side branches
Extremely angulated segments, ≥90 degrees	Degenerated vein grafts with friable lesions

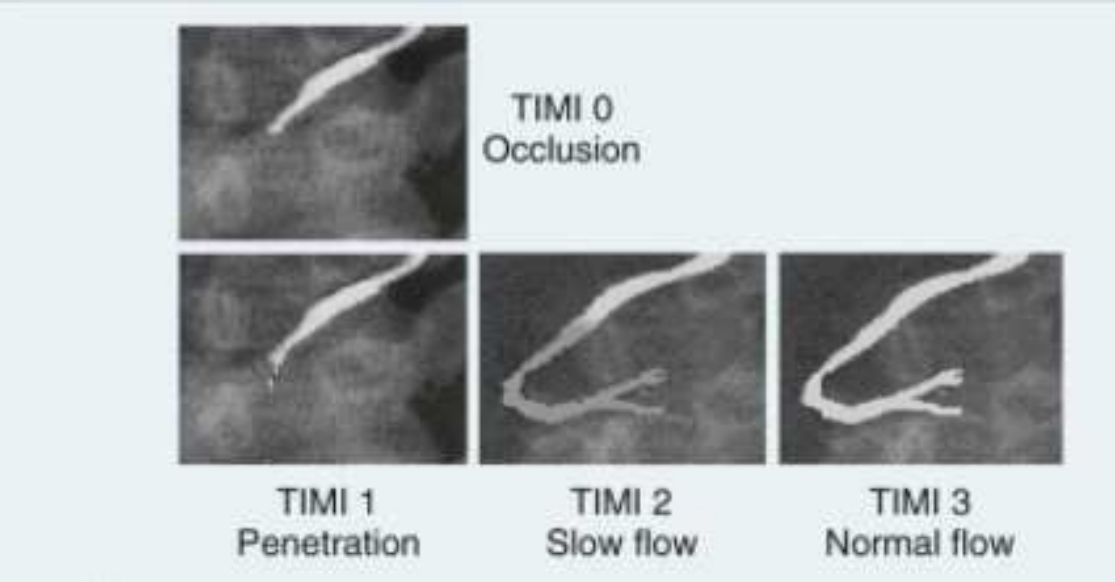
CORONARY COLLATERALS

RENTROP CLASSIFICATION:

- GRADE 0:No filling
- GRADE 1: Small side branches.
- GRADE 2:Partial epicardial filling of the coronary artery.
- GRADE 3:Complete epicardial filling of the coronary artery.

THROMBOLYSIS IN MYOCARDIAL INFARCTION (TIMI) FLOW:[95]

- GRADE0:No contrast flow through the stenosis.
- GRADE1: Flows through the stenosis but no opacification beyond obstruction
- GRADE2:Slow entry in to the terminal segment or delayed clearance
- GRADE3:Entry and clearance from stenosed segment similar to nonstenosed segment



TIMI FRAME COUNT:

Number of angiographic frames till the contrast arrives distal bed of vessel. Coronary blood flow ml/second—{21/observed TIMI frame count}X1.7

MYOCARDIAL NO REFLOW:

Reduced myocardial blood flow even after opening of an epicardial artery

MATERIALS AND METHODS

STUDY CENTRE:

Institute of Internal Medicine & Department of Cardiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

STUDY DESIGN:

Observational study (Prospective and Retrospective)

DURATION OF THE STUDY:

6 months

SAMPLE SIZE:

40 patients

INCLUSION CRITERIA:

- Patients with clinical features and ST Elevation myocardial infarction

EXCLUSION CRITERIA:

- Patients on drugs affecting platelet count and function
- Known coronary artery disease patients
- Unstable angina and non ST Elevation myocardial infarction patients

MATERIALS AND METHODS

DATA COLLECTION AND METHODS:

Patients admitted with ST elevation MI selected for clinical study as per inclusion / exclusion criteria were subjected to investigations like mean platelet volume, ECG, ECHO and coronary angiogram.

Mean platelet volume:

3ml of was taken in EDTA test tube and analyzed in Beckman Coulter LH780 analyzer to detect the mean platelet volume;

Mean platelet volume is the geometric mean of the transformed log normal platelet volume data in impedance technology system.

STASTICAL METHODS APPLIED:

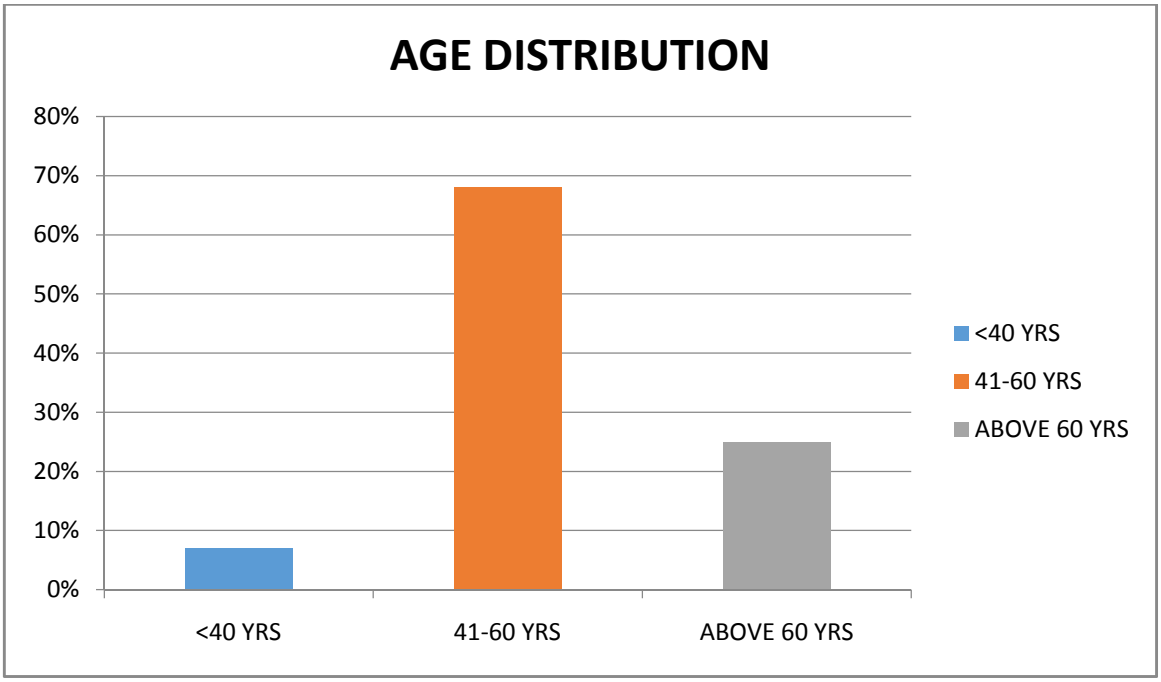
Datas were analysed using the SPSS software. Statistical significance was indicated by the Chi-square test. Variables were considered to be significant if $p<0.05$

OBSERVATION

OBSERVATION AND RESULTS

AGE DISTRIBUTION

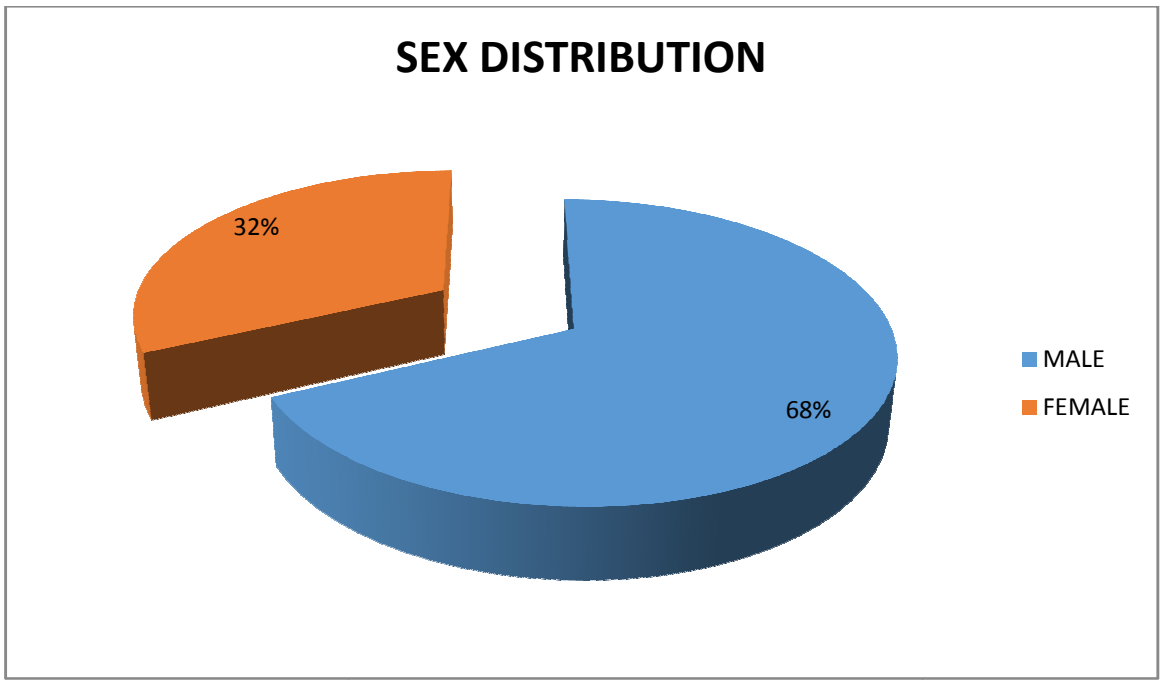
	Frequency	Percent
<40 YRS	3	7.5
41-60 YRS	27	67.5
ABOVE 60 YRS	10	25.0
Total	40	100.0



In our study, majority of the patients were in the age group of 41-60 years. (67.5%)

SEX DISTRIBUTION

	Frequency	Percent
MALE	27	67.5
FEMALE	13	32.5
Total	40	100.0



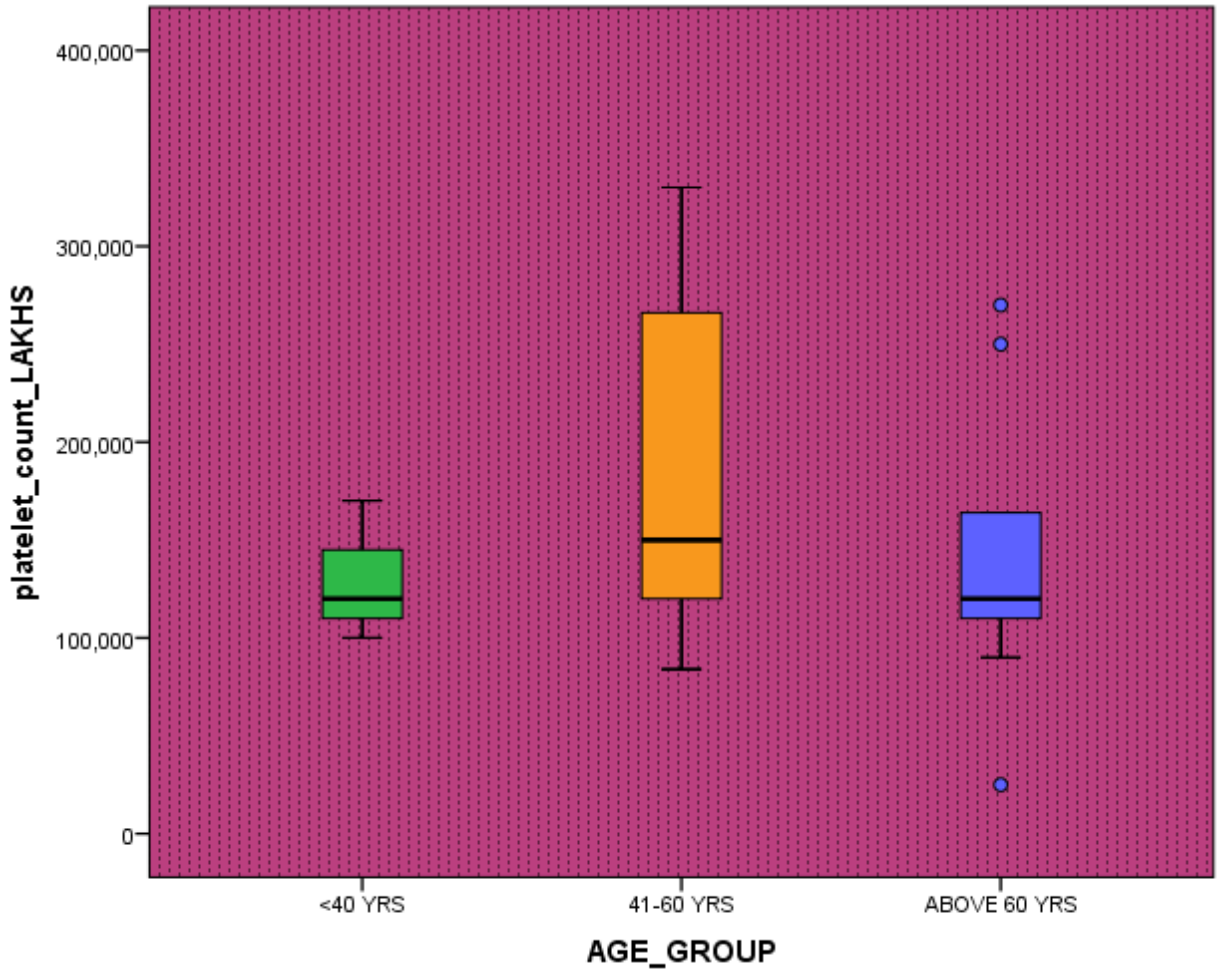
Out of 40 patients, 27 patients (67.5%) were males and 13 patients (32.5%) were females

AGE GROUP & PLATELET COUNT

		Mean	SD	Minimum	Maximum	Range
Platelet_count	<40 YRS	130000.00	36055.51	100000.00	170000.00	70000.00
	41-60 YRS	191333.33	86565.23	84000.00	330000.00	246000.00
	ABOVE 60 YRS	142500.00	72720.55	25000.00	270000.00	245000.00

In our study, the mean platelet count in patients <40 years was 1,30,000, in patients 41-60 years it was 1,91,333 and in patiens >60 years it was 1,42,500.

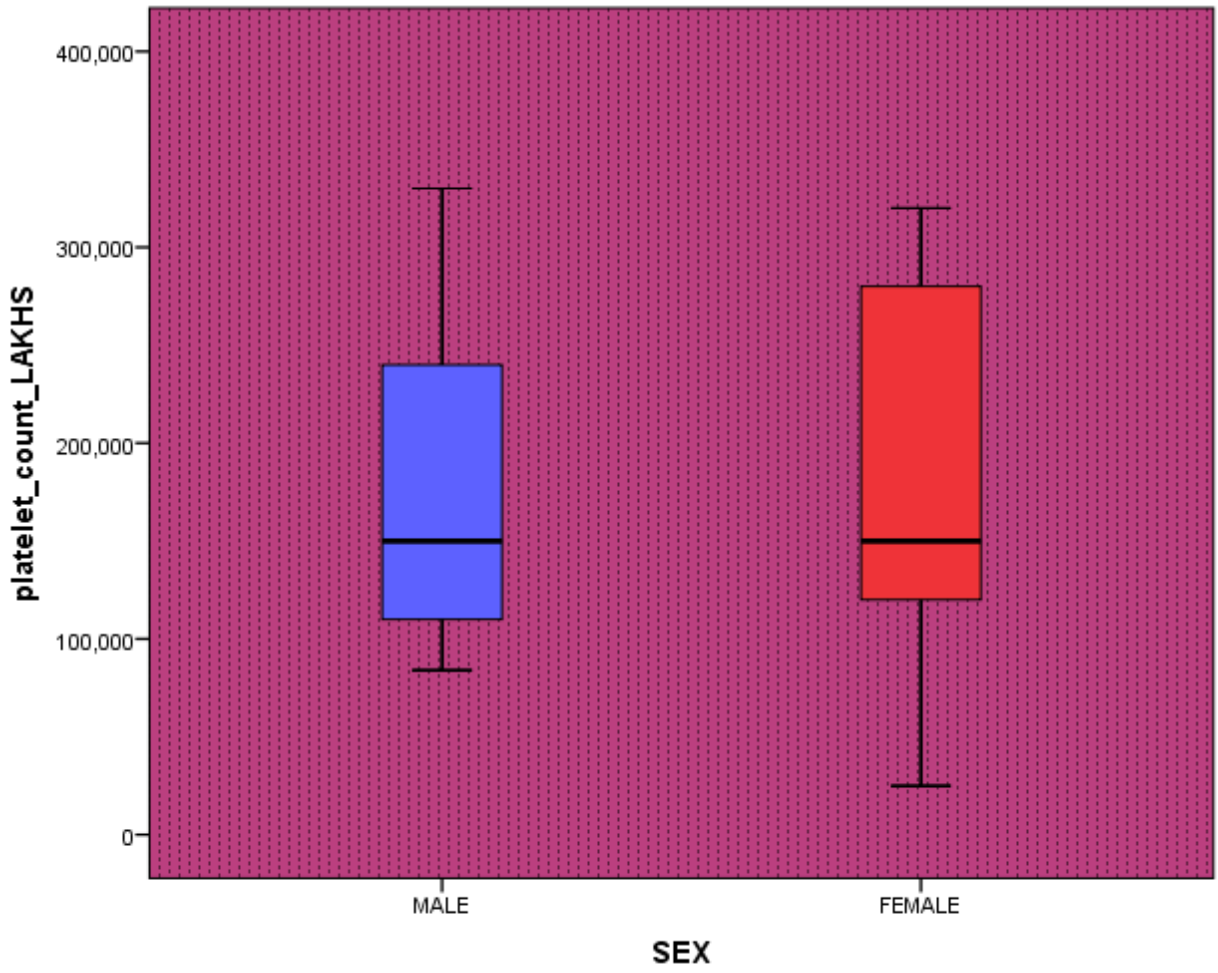
AGE GROUP & PLATELET COUNT



SEX & PLATELET COUNT

SEX		Mean	Std. Deviation	Minimum	Maximum	Range
platelet _count	MALE	170518.52	77959.74	84000.00	330000.00	246000.00
	FEMALE	182846.15	95526.48	25000.00	320000.00	295000.00

SEX AND PLATELET COUNT



DIABETES MELLITUS & MPV

			MPV		Total
			<9.5	>9.5	
	NORMAL	Count	14	7	21
		% within MPV	63.6%	38.9%	52.5%
	DM	Count	8	11	19
		% within MPV	36.4%	61.1%	47.5%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson Chi-square test: p value – 0.119

SYSTEMIC HYPERTENSION & MPV

			MPV		Total
			<9.5	>9.5	
	NORMAL	Count	16	10	26
		% within MPV	72.7%	55.6%	65.0%
	SHT	Count	6	8	14
		% within MPV	27.3%	44.4%	35.0%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson Chi-square test: p value – 0.257

SMOKING & MPV

			MPV		Total
			<9.5	>9.5	
SMOKING	NORMAL	Count	18	10	28
		% within MPV	81.8%	55.6%	70.0%
	YES	Count	4	8	12
		% within MPV	18.2%	44.4%	30.0%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson Chi-square test: p value – 0.071

ALCOHOL & MPV

			MPV		Total
			<9.5	>9.5	
Alcohol	NO	Count	18	10	28
		% within MPV	81.8%	55.6%	70.0%
	YES	Count	4	8	12
		% within MPV	18.2%	44.4%	30.0%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson Chi-square test: p value – 0.071

DISTRIBUTION OF TYPE OF MI

	Frequency	Percent
AWMI	12	30.0
EAWMI	14	35.0
IWMI	7	17.5
IWMI + RVMI	3	7.5
LWMI	3	7.5
LWMI IWMI	1	2.5
Total	40	100.0

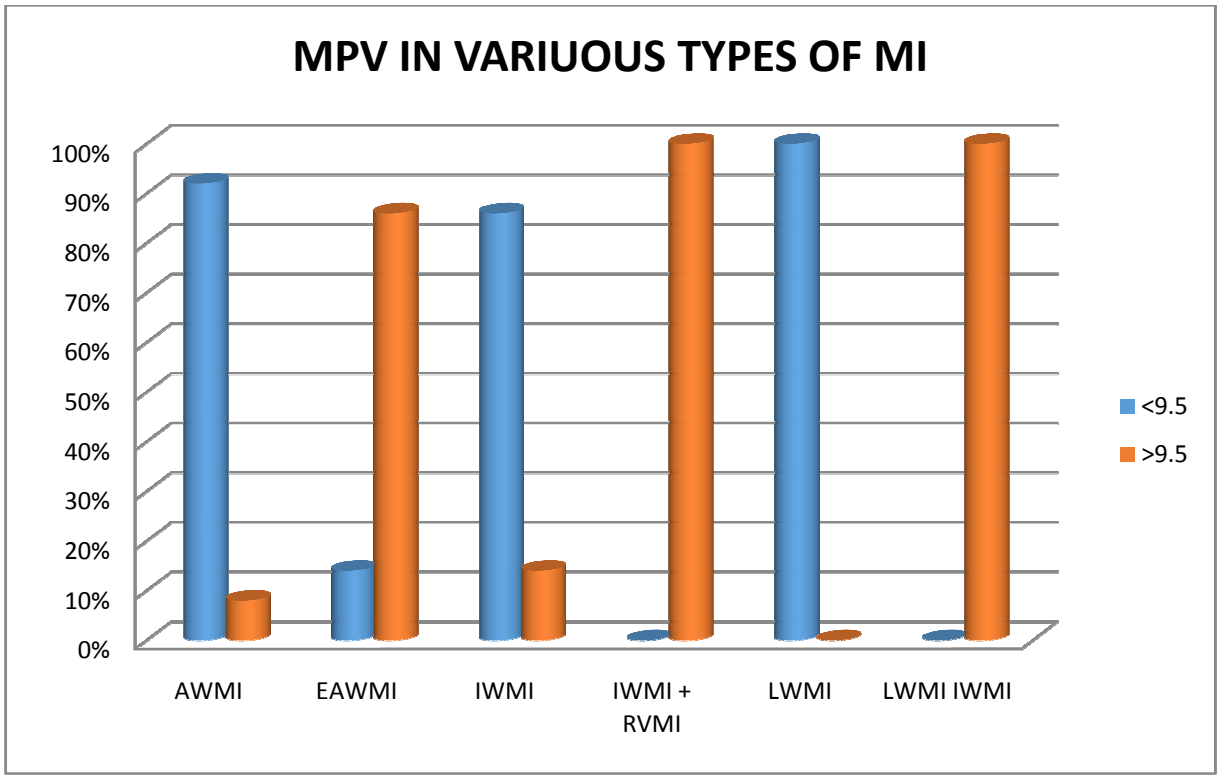
In our study, extensive anterior wall MI was the most common MI present in 14 patients (35%)

MPV IN TYPE OF MI

			Type of MI						Total
			AWMI	EAWMI	IWMI	IWMI + RVMI	LWMI	LWMI IWMI	
MPV	<9.5	Count	11	2	6	0	3	0	22
		% within IN	91.7%	14.3%	85.7%	0.0%	100.0%	0.0%	55.0%
	>9.5	Count	1	12	1	3	0	1	18
		% within IN	8.3%	85.7%	14.3%	100.0%	0.0%	100.0%	45.0%
Total		Count	12	14	7	3	3	1	40
		% within IN	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-square test: p value – 0.000

In our study, there was a significant correlation between MPV >9.5 and the infarct size which was statistically significant.

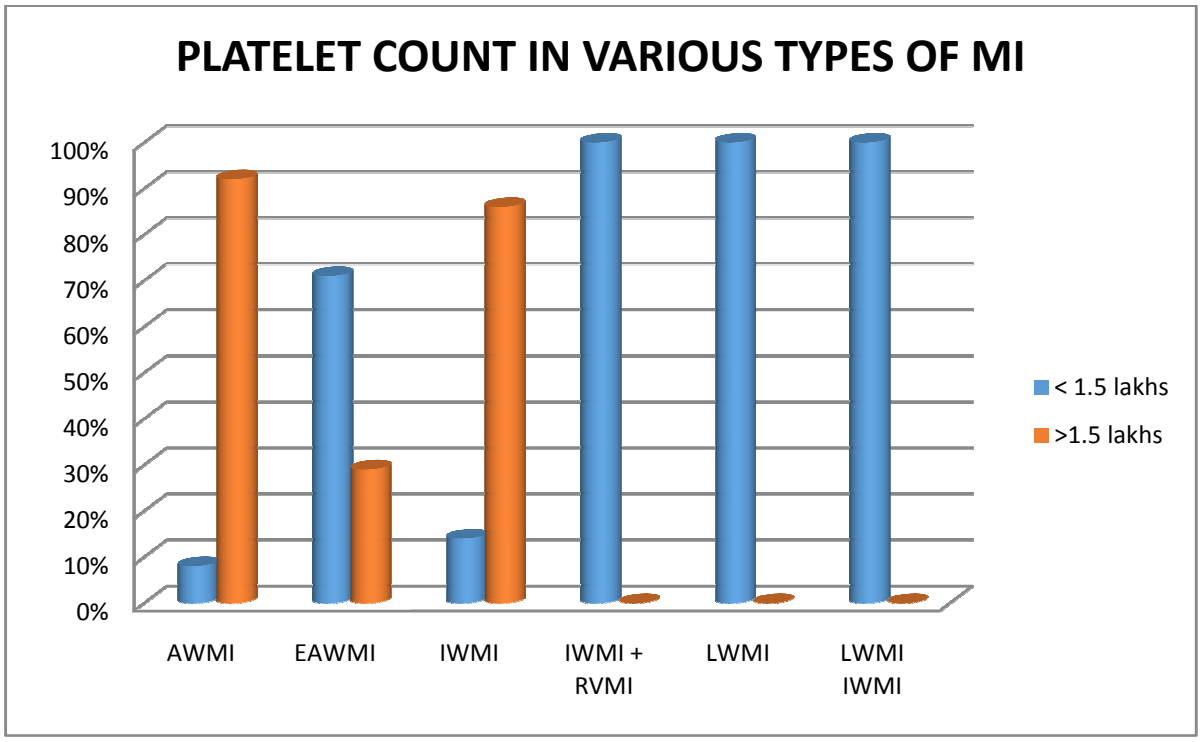


PLATELET COUNT IN VARIOUS TYPES OF MI

Platelet count		MI						Total
		AWMI	EAWMI	IWMI	IWMI + RVMI	LWMI	LWMI IWMI	
<1.5 lakhs	Count	1	10	1	3	3	1	19
	% within MI	8.3%	71.4%	14.3%	100.0%	100.0%	100.0%	47.5%
>1.5 lakhs	Count	11	4	6	0	0	0	21
	% within MI	91.7%	28.6%	85.7%	0.0%	0.0%	0.0%	52.5%
Total	Count	12	14	7	3	3	1	40
	% within MI	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0 %

Pearson Chi-square test: p value - 0.001

In our study, there was a significant correlation between low platelet count (<1.5 lakhs) and infarct size which was statistically significant.

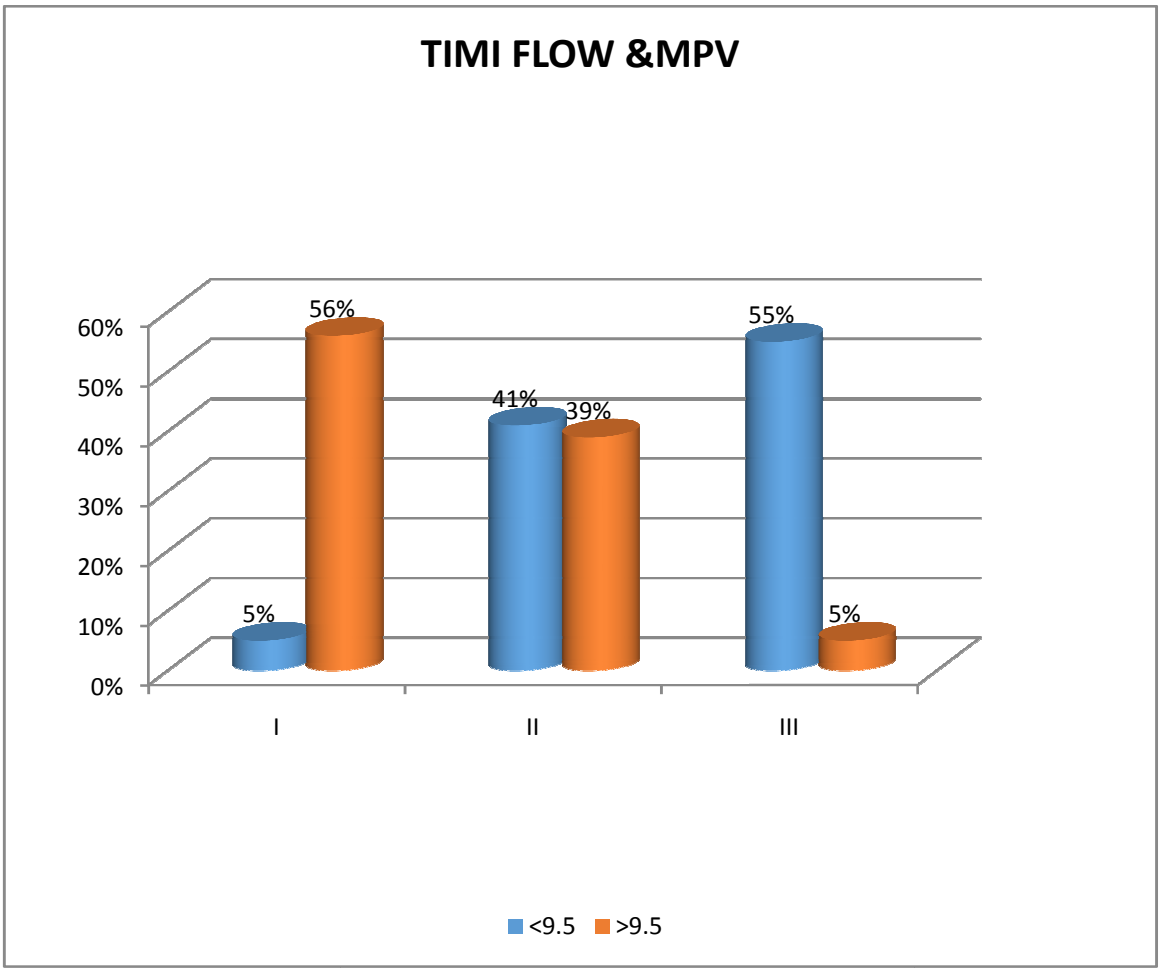

PLATELET COUNT IN VARIOUS TYPES OF MI
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
AWMI
EAWMI
IWMI
IWMI + RVMI
LWMI
LWMI IWMI
< 1.5 lakhs
>1.5 lakhs

TIMI FLOW AND MPV

			MPV		Total
			<9.5	**>9.5**	
TIMI FLOW	I	Count	1	10	11
		% within MPV	4.5%	55.6%	27.5%
	II	Count	9	7	16
		% within MPV	40.9%	38.9%	40.0%
	III	Count	12	1	13
		% within MPV	54.5%	5.6%	32.5%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson chi-square test p value – 0.000

In our study, there was a significant correlation between high mean platelet volume (>9.5) and low TIMI flow which was statistically significant

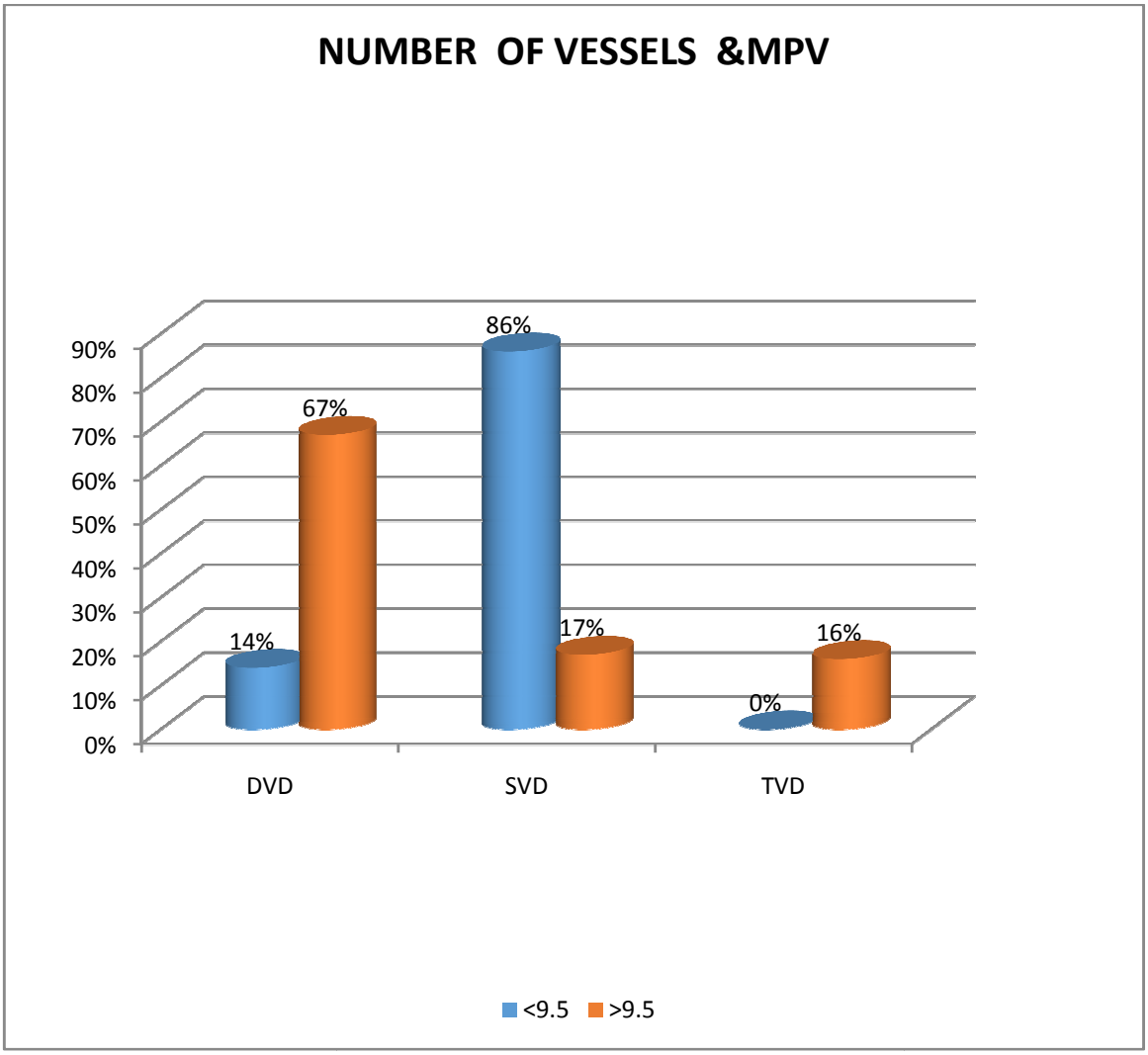

TIMI FLOW &MPV
60%
50%
40%
30%
20%
10%
0%
56%
5%
41%
39%
55%
5%
I
II
III
<9.5
>9.5

NUMBER OF VESSELS AND MPV

			MPV		Total
			<9.5	**>9.5**	
NO OF VESSELS	DVD	Count	3	12	15
		% within MPV	13.6%	66.7%	37.5%
	SVD	Count	19	3	22
		% within MPV	86.4%	16.7%	55.0%
	TVD	Count	0	3	3
		% within MPV	0.0%	16.7%	7.5%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson chi-square test p value – 0.000

In our study, as the number of infarcted vessels increase, there was a significant rise in mean platelet volume (>9.5) and this was statistically significant.

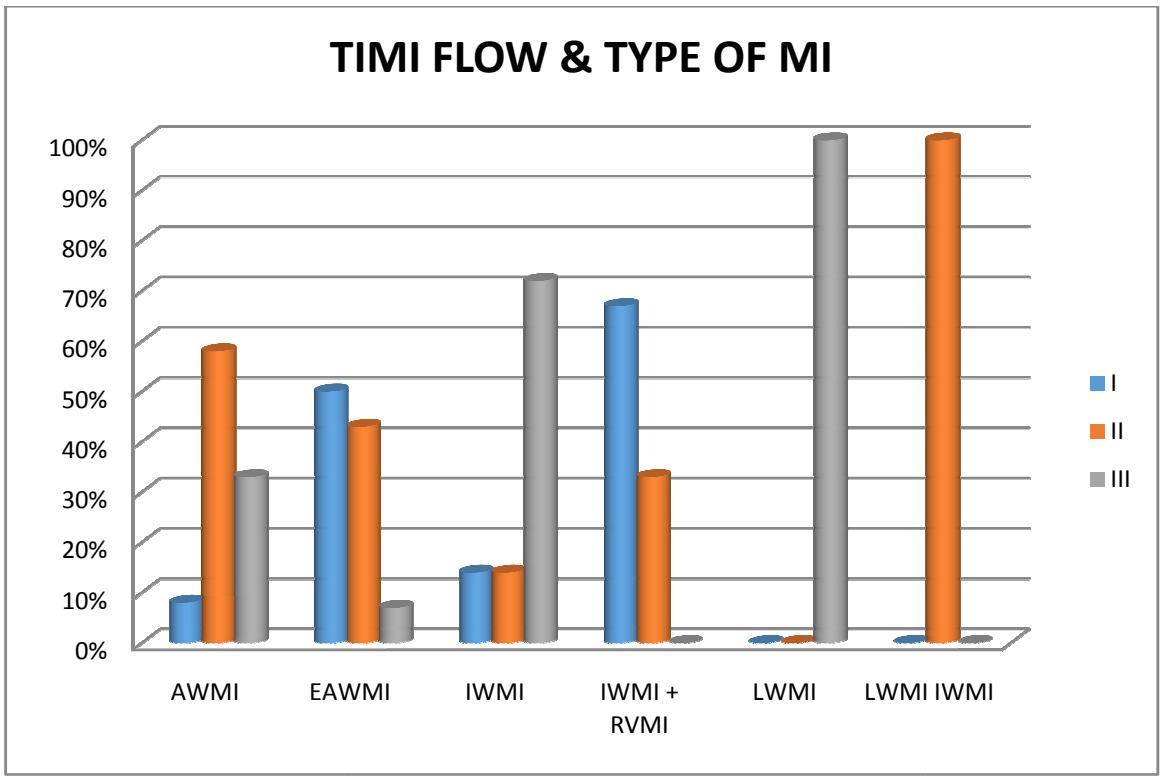

NUMBER OF VESSELS &MPV
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
14%
67%
86%
17%
0%
16%
DVD
SVD
TVD
<9.5
>9.5

TIMI FLOW & TYPE OF MI

			MI						Total
			AWMI	EAWMI	IWMI	IWMI + RVMI	LWMI	LWMI IWMI	
TIMI FLOW	I	Count	1	7	1	2	0	0	11
		% within MI	8.3%	50.0%	14.3%	66.7%	0.0%	0.0%	27.5%
	II	Count	7	6	1	1	0	1	16
		% within MI	58.3%	42.9%	14.3%	33.3%	0.0%	100.0%	40.0%
	III	Count	4	1	5	0	3	0	13
		% within MI	33.3%	7.1%	71.4%	0.0%	100.0%	0.0%	32.5%
Total		Count	12	14	7	3	3	1	40
		% within MI	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

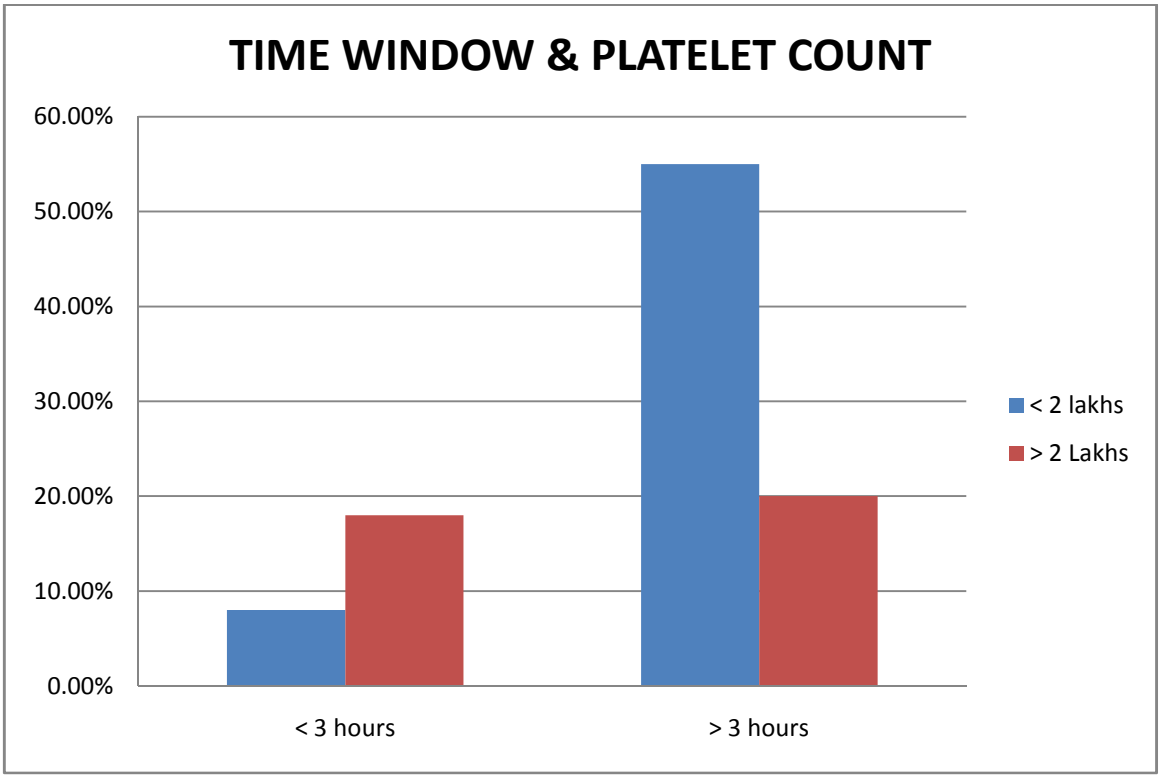
Pearson chi-square test p value – 0.010

As the infarct size increases there was low TIMI flow which was statistically significant in our study.

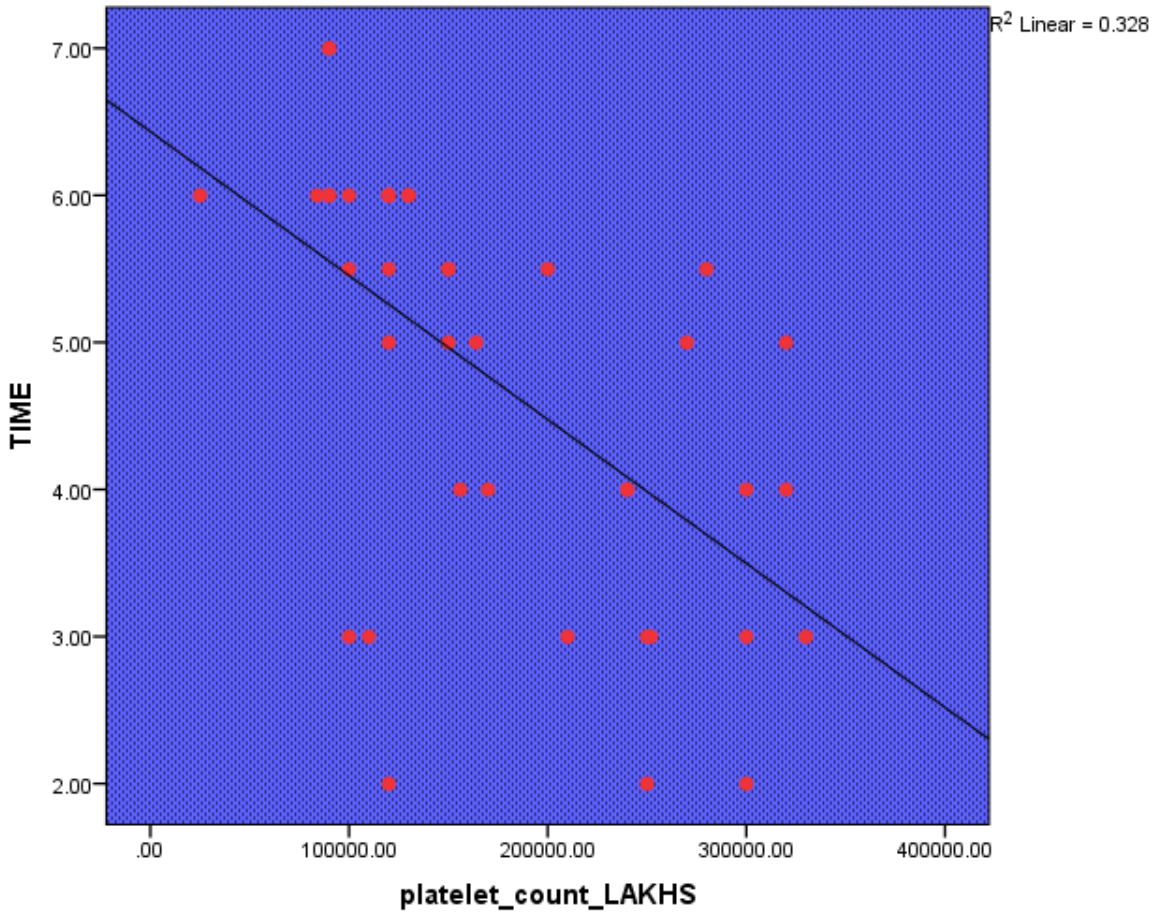

TIMI FLOW & TYPE OF MI
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
AWMI
EAWMI
IWMI
IWMI + RVMI
LWMI
LWMI IWMI
I
II
III

TIME WINDOW& PLATELET COUNT

TIME	PLATELET				Total			
	<2 LAKHS		>2 LAKHS					
	NO	%	NO	%	NO	%	R	P value
< 3 HOURS	3	12.0%	7	46.7%	10	25.0%	-0.388*	**0.013**
> 3 HOURS	22	88.0%	8	53.3%	30	75.0%		
Total	25	100.0%	15	100.0%	40	100.0%		



CORRELATION BETWEEN TIME WINDOW& PLATELET COUNT

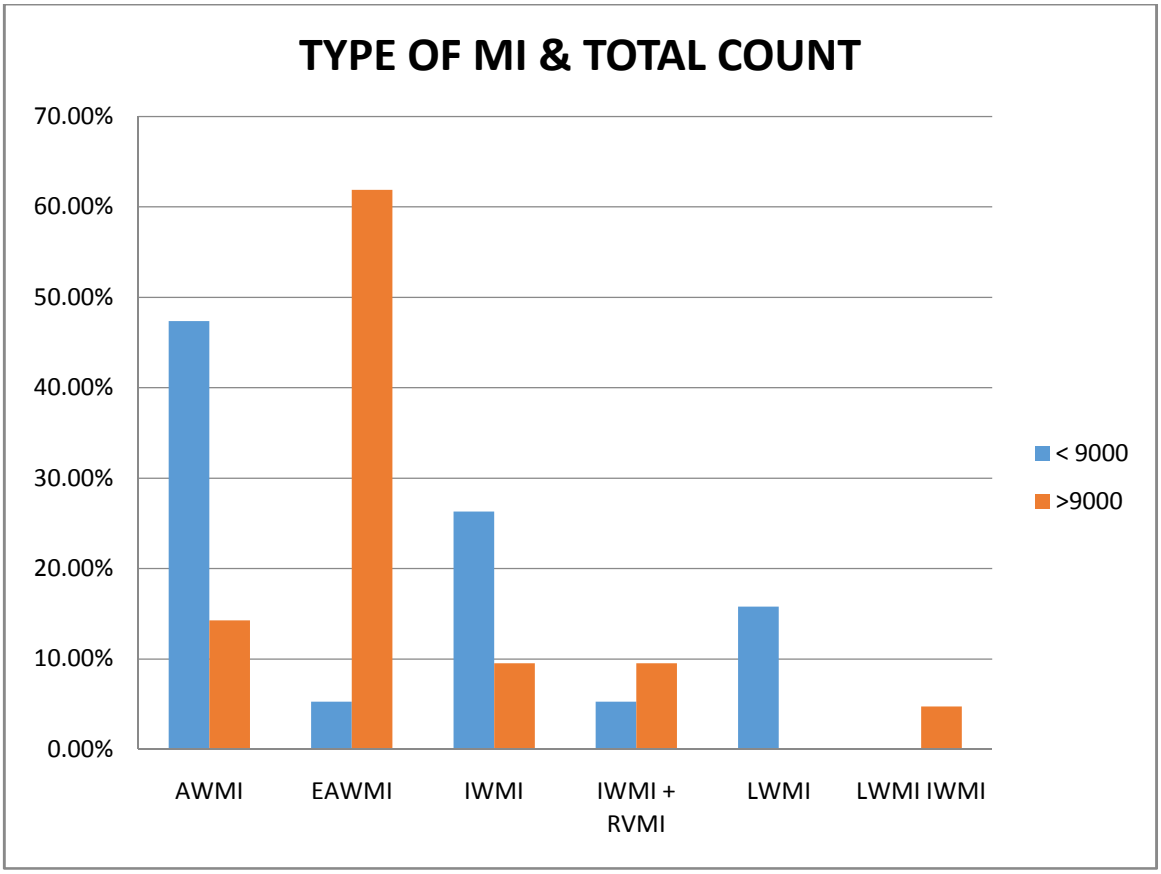


As the time interval to treatment increases, more platelets will be consumed and platelet count will be low which statistically significant in our study with a p value 0.013.

TYPE OF MI & TOTAL COUNT

		TOTAL COUNT				Total			
		< 9000		>9000					
		No.	**%**	**No.**	**%**	**No.**	**%**	**Chi square**	**P value**
MI TYPE	AWMI	9	22.5%	3	7.5%	12	30.0%	18.852*	**0.002**
	EAWMI	1	2.5%	13	32.5%	14	35.0%		
	LWMI	5	12.5%	2	5.0%	7	17.5%		
	LWMI+ RWMI	1	2.5%	2	5.0%	3	7.5%		
	LWMI	3	7.5%	0	0.0%	3	7.5%		
	LWMI IWMI	0	0.0%	1	2.5%	1	2.5%		
Total		19	47.5%	21	52.5%	40	100.0%		

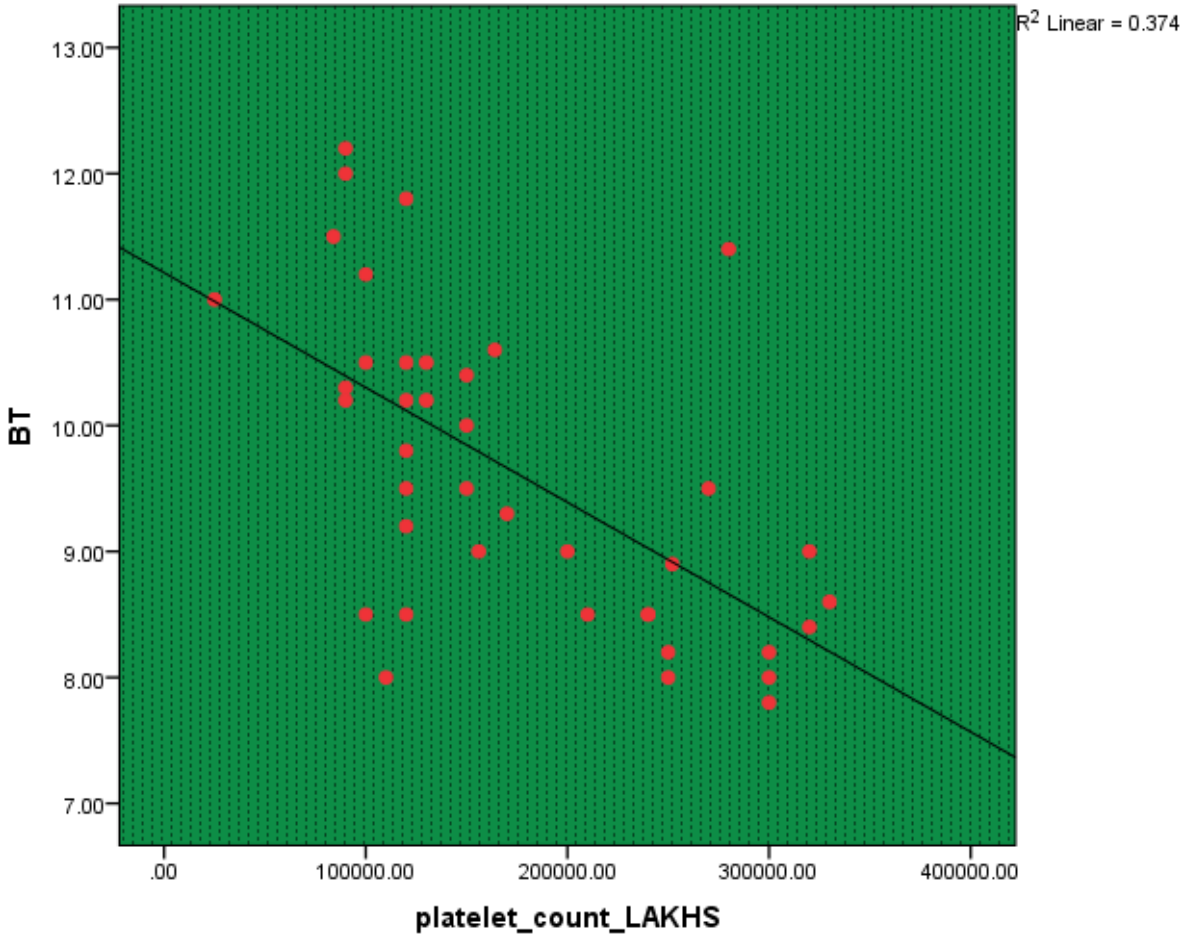
As the infarct size increases, there was increase in total count in our study which was statistically significant with a p value 0.002


TYPE OF MI & TOTAL COUNT
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
AWMI
EAWMI
IWMI
IWMI + RVMI
LWMI
LWMI IWMI
< 9000
>9000

MPV & PLATELET COUNT

		PLATELET				Total			
		<2 LAKHS		>2 LAKHS					
		NO	%	NO	%	NO	%	r	P value
MPV	<9.5	8	20.0%	14	35.0%	22	55.0%	-0.597*	0.000
	>9.5	17	42.5%	1	2.5%	18	45.0%		
Total		25	62.5%	15	37.5%	40	100.0%		

CORRELATION BETWEEN MPV & PLATELET COUNT



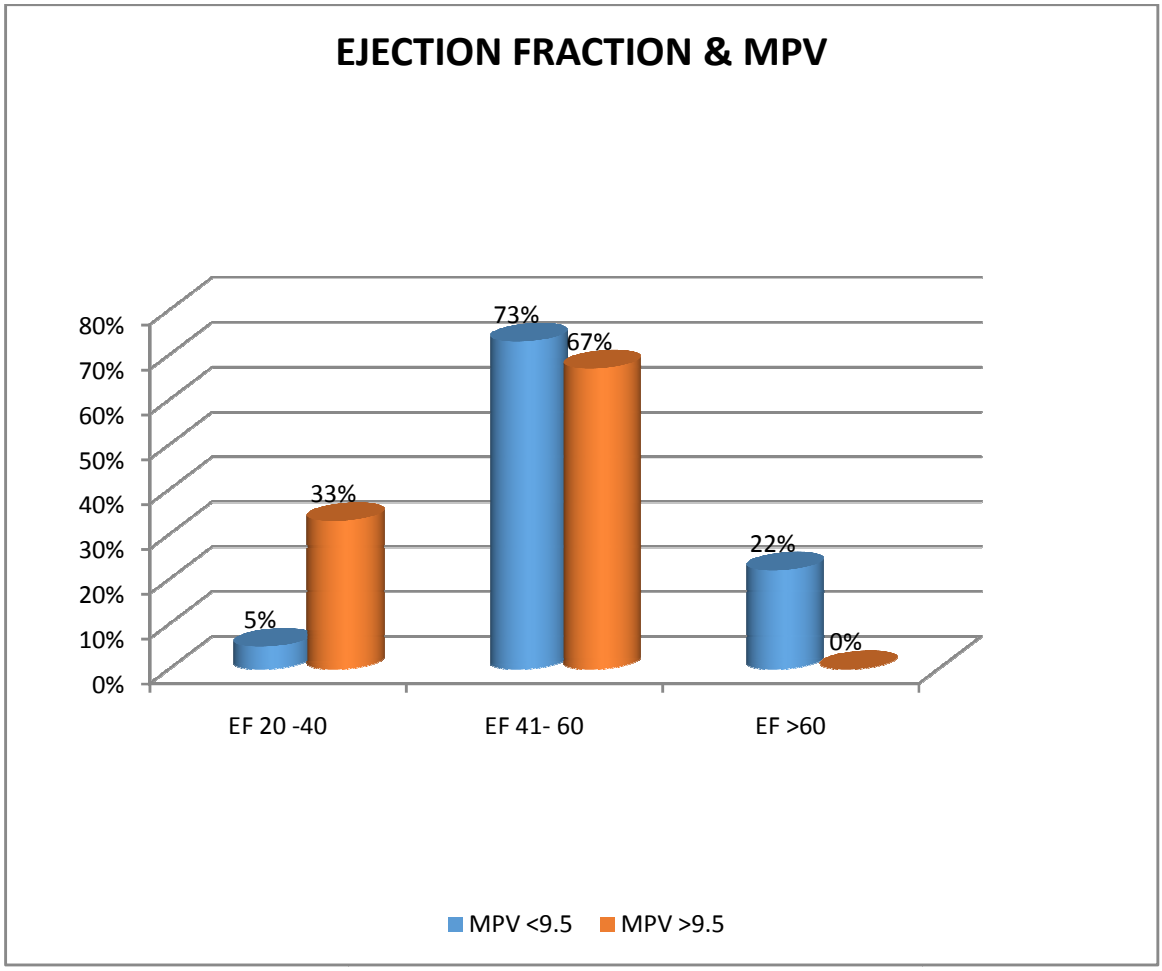
In our study, as the mean platelet volume increases there was a decrease in platelet count which was statistically significant with a p value 0.000

EF CLASS & MPV

			MPV		Total
			<9.5	**>9.5**	
EF CLASS	20-40	Count	1	6	7
		% within MPV	4.5%	33.3%	17.5%
	41-60	Count	16	12	28
		% within MPV	72.7%	66.7%	70.0%
	ABOVE 60	Count	5	0	5
		% within MPV	22.7%	0.0%	12.5%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

Pearson chi-square test p value – 0.012

In our study, when there was high mean platelet volume, there was low ejection fraction which was statistically significant.


EJECTION FRACTION & MPV
80%
70%
60%
50%
40%
30%
20%
10%
0%
5%
33%
73%
67%
22%
0%
EF 20 -40
EF 41- 60
EF >60
MPV <9.5
MPV >9.5

INFARCT RELATED ARTERY AND MPV

			MPV		Total
			<9.5	>9.5	
IRA	DISTAL LAD	Count	3	0	3
		% within MPV	13.6%	0.0%	7.5%
	DISTAL RCA	Count	4	0	4
		% within MPV	18.2%	0.0%	10.0%
	DISTALRCA	Count	1	0	1
		% within MPV	4.5%	0.0%	2.5%
	LCX	Count	3	0	3
		% within MPV	13.6%	0.0%	7.5%
	PROXIMAL LAD	Count	8	0	8
		% within MPV	36.4%	0.0%	20.0%
	PROXIMAL LAD + PROXIMAL LCX DISTAL RCA	Count	0	1	1
		% within MPV	0.0%	5.6%	2.5%
	PROXIMAL LAD DISTAL LCX	Count	0	2	2
		% within MPV	0.0%	11.1%	5.0%
	PROXIMAL LAD LCX	Count	2	7	9
		% within MPV	9.1%	38.9%	22.5%
	PROXIMAL LAD MID LCX MID RCA	Count	0	1	1
		% within MPV	0.0%	5.6%	2.5%
	PROXIMAL LAD PROXIMAL LCX DISTAL RCA	Count	0	1	1
		% within MPV	0.0%	5.6%	2.5%
	PROXIMAL LAD RCA	Count	0	1	1
		% within MPV	0.0%	5.6%	2.5%
	PROXIMAL RCA	Count	1	1	2
		% within MPV	4.5%	5.6%	5.0%
	PROXIMAL RCA LCX	Count	0	2	2
		% within MPV	0.0%	11.1%	5.0%
	PROXIMALRCA	Count	0	2	2
		% within MPV	0.0%	11.1%	5.0%
Total		Count	22	18	40
		% within MPV	100.0%	100.0%	100.0%

In our study, when there was high mean platelet volume, the most common site of lesion was in proximal LAD + proximal LCX followed by proximal LAD + distal LCX which was statistically significant with a p value 0.003.

DISCUSSION

DISCUSSION

Our study was conducted in Institute of Internal Medicine and Department of cardiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. 40 patients fulfilling the inclusion and exclusion criteria were included in our study. Following observations were made from our study.

Age distribution:

Majority of cases were in the age group of 41-60 years with 27 patients (67.5%). This was similar to the study of Mehmet Bilje et al.

Sex distribution:

Out of 40 patients, 27 patients (67.5%) were males and 13 patients (32.5%) were females. Male to female ratio was 2:1

Age group and platelet count:

The mean platelet count in patients <40 years was 1,30,000, in patients 41-60 years it was 1,91,333 and in patiens >60 years it was 1,42,500.

Mean platelet volume and Diabetes & Hypertension:

No significant correlation was observed in our study between mean platelet volume and Diabetes mellitus.

Similarly there was no significant correlation between mean platelet volume and systemic hypertension. This was similar to the results from Bath et al (1996)

Mean platelet volume and Smoking & alcoholism

There was no significant correlation observed between mean platelet volume and smoking & alcoholism in this study.

Distribution of various types of MI:

Extensive anterior wall MI was the most common MI present in 14 patients (35%), followed by anterior wall MI in 12 patients (30%) and inferior wall MI 7 patients (17.5%).

Mean platelet volume and infarct size:

There was a significant correlation between MPV >9.5 and the infarct size which was statistically significant (p value 0.000). As the MPV increases, the infarct size also increases. This observation was similar to the study of Morell et al. in which he correlated admission mean platelet volume to the infarct size assessed by cardiac MRI.

Platelet count and infarct size:

There was a significant correlation between low platelet count (<1.5 lakhs) and infarct size which was statistically significant. (p value – 0.001).

With decreasing platelet count, the infarct size increases. This was similar to the study of Mehmet Bilje et al.

TIMI flow and Mean platelet volume:

There was a significant correlation between high mean platelet volume (>9.5) and low TIMI flow which was statistically significant (p value 0.000) which was similar to the study of Opolski G et al.

Number of vessels and Mean platelet volume:

As the number of infarcted vessels increase, there was a significant rise in mean platelet volume (>9.5) and this was statistically significant (p value – 0.000). This was similar to the study of Murat Cayli et al.

TIMI flow and type of MI:

In this study there was a significant correlation between TIMI flow and type of MI which statistically significant (p value – 0.010). As the area of infarction increases there was low TIMI flow.

Time window and Platelet count:

As the time interval to treatment increases, more platelets will be consumed and platelet count will be low which statistically significant in study this with a p value 0.013.

Type of MI and total count:

In this study there was an increase in total count as the infarct size increases which was statistically significant (p value 0.002). This was similar to the observations made by James Scheuer et al.

MPV and Platelet count:

As the mean platelet volume increases there was a decrease in platelet count in this study which was statistically significant (p value 0.000)

Ejection fraction and MPV:

Significant correlation was observed in this study between MPV and ejection fraction. When there was high mean platelet volume, there was low ejection fraction which was statistically significant in our study (p value – 0.012). This observation was similar to that of Celik S et al.

CONCLUSION

CONCLUSIONS

- Admission mean platelet volume represents the reactivity of platelets – thrombus burden.
- High admission mean platelet volume has significant impact on prognosis of myocardial infarction.
- Admission mean platelet volume was not affected by diabetes mellitus, systemic hypertension, smoking, alcohol intake, age and sex.
- Infarct size was directly proportional to admission mean platelet volume.
- Successfulness of thrombolysis was inversely proportional to admission mean platelet volume.
- Infarct related artery patency and TIMI flow were inversely proportional to admission mean platelet volume
- Platelet count was inversely proportional to admission MPV.
- Total count has correlated with infarct size.

BIBLIOGRAPHY

1. Thygesen K, Alpert JS, White HD, et al: Universal definition of myocardial infarction. *Circulation* 2007; 116:2634
2. Hochholzer W, Buettner HJ, Trenk D, et al: New definition of myocardial infarction: Impact on long-term mortality. *Am J Med* 2008; 121:399.
3. *The heart. In Kumar V, Abbas AK, Fausto N, Aster J [eds]: Robbins and Cotran Pathologic Basis of Disease. 8th ed. Philadelphia, WB Saunders, 2010, pp 529-587.)*
4. Funaro S, La Torre G, Madonna M, et al: Incidence, determinants, and prognostic value of reverse left ventricular remodelling after primary percutaneous coronary intervention: results of the Acute Myocardial Infarction Contrast Imaging (AMICI) multicenter study. *Eur Heart J* 2009; 30:566.
5. Schuster EH, Bulkley BH: Ischemia at a distance after acute myocardial infarction: A cause of early postinfarction angina. *Circulation* 1980; 62:509
6. DeWood MA, Spores J, Notske R, et al: Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. *N Engl J Med* 1980; 303:897
7. Chua S, Chang LT, Sun CK, et al: Time courses of subcellular signal transduction and cellular apoptosis in remote viable myocardium of rat left ventricles following acute myocardial infarction: Role of pharmacomodulation. *J Cardiovasc Pharmacol Ther* 2009; 14:104
8. Schoen FJ: *The heart*. In: Kumar V, Abbas AK, Fausto N, ed. *Robbins & Cotran Pathologic Basis of Disease*, 8th ed. Philadelphia: WB Saunders; 2010:

9. Vargas SO, Sampson BA, and Schoen FJ: Pathologic detection of early myocardial infarction: A critical review of the evolution and usefulness of modern techniques. *Mod Pathol* 1999; 12:635.
10. Pasotti M, Prati F, Arbustini E: The pathology of myocardial infarction in the pre- and post-interventional era. *Heart* 2006; 92:1552
11. Noel TE, Kontos MC: *Troponin and other markers of necrosis for risk stratification in patients with acute coronary syndromes*. In: de Lemos JA, ed. *Biomarkers in Heart Disease*, Oxford: Blackwell Publishing; 2008:22-39.
12. Popescu BA, Antonini-Canterin F, Temporelli PL, et al: Right ventricular functional recovery after acute myocardial infarction: Relation with left ventricular function and interventricular septum motion. GISSI-3 echo substudy. *Heart* 2005; 91:484.
13. Tjandrawidjaja MC, Fu Y, Kim DH, et al: Compromised atrial coronary anatomy is associated with atrial arrhythmias and atrioventricular block complicating acute myocardial infarction. *J Electrocardiol* 2005; 38:271.
14. Schuster EH, Bulkley BH: Ischemia at a distance after acute myocardial infarction: A cause of early postinfarction angina. *Circulation* 1980; 62:509.
15. Funaro S, La Torre G, Madonna M, et al: Incidence, determinants, and prognostic value of reverse left ventricular remodeling after primary percutaneous coronary intervention: results of the Acute Myocardial Infarction Contrast Imaging (AMICI) multicenter study. *Eur Heart J* 2009; 30:566.
16.. Hochman JS: Cardiogenic shock complicating acute myocardial infarction: Expanding the paradigm. *Circulation* 2003; 107:2998
17. Ruan W, Lu L, Zhang Q, et al: Serial assessment of left ventricular remodeling and function by echo-tissue Doppler imaging after myocardial

infarction in streptozotocin-induced diabetic swing. *J Am Soc Echocardiogr* 2009; 22:530

18. Weisman HF, Bush DE, Mannisi JA, et al: Cellular mechanisms of myocardial infarct expansion. *Circulation* 1988; 78:186
19. Konstam MA: Patterns of ventricular remodeling after myocardial infarction: Clues toward linkage between mechanism and morbidity. *JACC Cardiovasc Imaging* 2008; 1:592.
20. *Braunwald E, Pfeffer MA: Ventricular enlargement and remodeling following acute myocardial infarction: Mechanisms and management. Am J Cardiol 68:4D, 1991*
21. Bodis J, Boncz I, Kriszbacher I: Permanent stress may be the trigger of an acute myocardial infarction on the first work-day of the week. *Int J Cardiol* 2009
22. Assali AR, Brosh D, Vaknin-Assa H, et al: The impact of circadian variation on outcomes in emergency acute anterior myocardial infarction percutaneous coronary intervention. *Catheter Cardiovasc Interv* 2006; 67:221
23. Leiza JR, de Llano JM, Messa JB, et al: New insights into the circadian rhythm of acute myocardial infarction in subgroups. *Chronobiol Int* 2007; 24:129
24. Feringa HH, Karagiannis SE, Vidakovic R, et al: The prevalence and prognosis of unrecognized myocardial infarction and silent myocardial ischemia in patients undergoing major vascular surgery. *Coron Artery Dis* 2007; 18:571
25. Laukkanen A, Ikaheimo M, Luukinen H: Practices of clinical examination of heart failure patients in primary health care. *Cent Eur J Public Health* 2006; 14:86

26. Vukanovic-Criley JM, Criley S, Warde CM, et al: Competency in cardiac examination skills in medical students, trainees, physicians, and faculty: A multicenter study. *Arch Intern Med* 2006; 166:610
27. The Criteria Committee of the New York Heart Association: *Nomenclature and Criteria for Diagnosis*. 9th ed. Boston, Little Brown, 1994
28. Fonarow GC, Adams Jr KF, Abraham WT, et al: Risk stratification for in-hospital mortality in acutely decompensated heart failure: Classification and regression tree analysis. *JAMA* 2005; 293:572
29. March SK, Bedynek Jr JL, Chizner MA: Teaching cardiac auscultation: Effectiveness of a patient-centered teaching conference on improving cardiac auscultatory skills. *Mayo Clin Proc* 2005; 80:1443
30. Tavel ME: Cardiac auscultation: A glorious past—and it does have a future! *Circulation* 2006; 113:1255
31.. Killip 3rd T, Kimball JT: Treatment of myocardial infarction in a coronary care unit. A two-year experience with 250 patients. *Am J Cardiol* 1967; 20:457
32. Antman EM: Decision making with cardiac troponin tests. *N Engl J Med* 2002; 346:2079
33. Jaffe AS, Babuin L, and Apple FS: Biomarkers in acute cardiac disease: The present and the future. *J Am Coll Cardiol* 2006; 48:1
34. Morrow DA, Cannon CP, Jesse RL, et al: National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: Clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Circulation* 2007; 115:e356
35. Kligfield P, Gettes L, Bailey JJ, et al: Recommendations for the standardization and interpretation of the electrocardiogram. Part I: The

electrocardiogram and its standardization. *J Am Coll Cardiol* 2007; 49:1109.

36. MacLeod R, Kornreich F, van Oosterom A, et al: Report of the first virtual visualization of the reconstructed electrocardiographic display symposium. *J Electrocardiol* 2005; 38:385.
37. Bacharova L, Selvester RH, Engblom H, and Wagner GS: Where is the central terminal located? In search of understanding the use of the Wilson central terminal for production of 9 of the 12 electrocardiogram leads. *J Electrocardiol* 2005; 38:119
38. *Modified from Goldberger AL: Myocardial Infarction: Electrocardiographic Differential Diagnosis. 4th ed. St. Louis, Mosby-Year Book, 1991.)*
39. *.(Modified from Goldberger AL: Clinical Electrocardiography: A Simplified Approach. 6th ed. St. Louis, CV Mosby, 1999*
40. *Modified from Wellens HJ: The value of the right precordial leads of the electrocardiogram. N Engl J Med 340:381, 1999; and Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines [Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction]. Circulation 110:e82, 2004.)*
41. Oh JK, Seward JB, Tajik AJ: *The Echo Manual*. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006
42. Mulvagh SL, Rakowski H, Vannan MA, et al: American Society of Echocardiography consensus statement on the clinical applications of ultrasonic contrast agents in echocardiography. *J Am Soc Echocardiogr* 2008; 21:1179

43. *Modified from Oh JK, Seward JB, And Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission of Mayo Foundation for Medical Education and Research.* ***B*** *modified from Lang RM, Bierig M, Devereux RB, et al: Recommendations for chamber quantification: A report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 18:1440, 2005. Used with permission*

44. Hanekom L, Jenkins C, Jeffries L, et al: Incremental value of strain rate analysis as an adjunct to wall-motion scoring for assessment of myocardial viability by dobutamine echocardiography: A follow-up study after revascularization. *Circulation* 2005; 112:3892.

45. Vartdal T, Brunvand H, Pettersen E, et al: Early prediction of infarct size by strain Doppler echocardiography after coronary reperfusion. *J Am Coll Cardiol* 2007; 49:1715.

46. Vignon P: Hemodynamic assessment of critically ill patients using echocardiography Doppler. *Curr Opin Crit Care* 2005; 11:227

47. Bursi F, Enriquez-Sarano M, Jacobsen SJ, and Roger VL: Mitral regurgitation after myocardial infarction: A review. *Am J Med* 2006; 119:103.

48. *From Oh JK, Seward JB, Tajik AJ: The Echo Manual. 3rd ed. Philadelphia, Lippincott Williams & Wilkins, 2006. Used with permission from Mayo Foundation for Medical Education and Research.)*

49. *Modified from Antman EM, Anbe DT, Armstrong PW, et al: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction: A report of the American College of Cardiology/American Heart*

Association Task Force on Practice Guidelines [Committee to Revise the 1999 Guidelines for the Management of Patients with Acute Myocardial Infarction]. Circulation 110:e82,

50. Le May MR, So DY, Dionne R, et al: A citywide protocol for primary PCI in ST-segment elevation myocardial infarction. *N Engl J Med* 2008; 358:231.

51.. Wiviott SD, Morrow DA, Frederick PD, et al: Performance of the thrombolysis in myocardial infarction risk index in the National Registry of Myocardial Infarction-3 and -4: A simple index that predicts mortality in ST-segment elevation myocardial infarction. *J Am Coll Cardiol* 2004; 44:783.

52. *(Modified from Kushner FG, Hand M, Smith SC Jr, et al: 2009 Focused Updates: ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction (updating the 2004 Guideline and 2007 Focused Update) and ACC/AHA/SCAI Guidelines on Percutaneous Coronary Intervention (updating the 2005 Guideline and 2007 Focused Update): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 120:2271, 2009.)*

53. Wiviott SD, Morrow DA, Frederick PD, et al: Performance of the thrombolysis in myocardial infarction risk index in the National Registry of Myocardial Infarction-3 and -4: A simple index that predicts mortality in ST-segment elevation myocardial infarction. *J Am Coll Cardiol* 2004; 44:783.

54. Morrow DA: *Heparin and low-molecular-weight heparin*. In: Manson JE, Buring JE, Ridker PM, Gaziano JM, ed. *Clinical Trials in Heart Disease: A Companion to Braunwald's Heart Disease,* 2nd ed. Philadelphia: Elsevier Saunders; 2004:45-65

55. Stone GW, Witzenbichler B, Guagliumi G, et al: Bivalirudin during primary PCI in acute myocardial infarction. *N Engl J Med* 2008; 358:2218.

56. Yusuf S, Mehta SR, Chrolavicius S, et al: Effects of fondaparinux on mortality and reinfarction in patients with acute ST-segment elevation myocardial infarction: The OASIS-6 randomized trial. *JAMA* 2009; 295:1519

57. Holmes Jr DR, Kereiakes DJ, Kleiman NS, et al: Combining antiplatelet and anticoagulant therapies. *J Am Coll Cardiol* 2009; 54:95

58. Ross R: Atherosclerosis-an inflammatory disease. N Engl J Med 340:115, 1999. [PMID: 9887164]

59. Harrison DG, Cai H: Endothelial control of vasomotion and nitric oxide production. *Cardiol Clin* 21:289, 2003. [PMID: 14621446]

60. Creager MA, Gallagher SJ, Girerd XJ, et al: L-arginine improves endothelium-dependent vasodilation in hypercholesterolemic humans. J Clin Invest 90:1248, 1992. [PMID: 1401062]

61. Spieker LE, Sudano I, Hurlimann D, et al: High-density lipoprotein restores endothelial function in hypercholesterolemic men. Circulation 105:1399, 2002. [PMID: 11914243]

62. Cybulsky MI, Gimbrone MA Jr: Endothelial expression of a mononuclear leukocyte adhesion molecule during atherogenesis. *Science* 251:788, 1991. [PMID: 1990440]

63. Gosling J, Slaymaker S, Gu L, et al: MCP-1 deficiency reduces susceptibility to atherosclerosis in mice that overexpress human apolipoprotein B. J Clin Invest 103:773, 1999. [PMID: 10079097]

64. Sugiyama S, Okada Y, Sukhova GK, et al: Macrophage myeloperoxidase regulation by granulocyte macrophage colony-stimulating factor in human atherosclerosis and implications in acute coronary syndromes. Am J Pathol 158:879, 2001. [PMID: 11238037]

65.(Adapted from S Kinlay, AP Selwyn, and P Libby: Inflammation, the endothelium, and the acute coronary syndromes. *J Cardiovasc Pharmacol* 32[suppl 3]:S62, 1998.)

66.Gonzalez MA, Selwyn AP: Endothelial function, inflammation, and prognosis in cardiovascular disease. *Am J Med* 115(suppl 8A):99S, 2003.

67.Stary HC: Natural history and histological classification of atherosclerotic lesions: An update. Arterioscler Thromb Vasc Biol 20:1177, 2000. [PMID: 10807728]

68.Naghavi M, Libby P, Falk E, et al: From vulnerable plaque to vulnerable patient: A call for new definitions and risk assessment strategies: Part II. Circulation 108:1772, 2003. [PMID: 14557340]

69.Casscells W, Naghavi M, Willerson JT: Vulnerable atherosclerotic plaque: A multifocal disease. *Circulation* 107:2072, 2003. [PMID: 12719287

70.Falk E: Plaque rupture with severe pre-existing stenosis precipitating coronary thrombosis. Characteristics of coronary atherosclerotic plaques underlying fatal occlusive thrombi. *Br Heart J* 50:127, 1983. [PMID

71.Karnicki K, Owen WG, Miller RS, McBane RD: Factors contributing to individual propensity for arterial thrombosis. *Arterioscler Thromb Vasc Biol* 22:1495, 2002. [PMID: 12231572]

72.Stenberg PE, Levin J: Mechanisms of platelet production. *Blood Cells* 15:23, 1989. [PMID: 2649182

73.Vitrat N, Cohen-Solal K, Pique C, et al: Endomitosis of human megakaryocytes are due to abortive mitosis. *Blood* 91:3711, 1998. [PMID: 9573008]

74.Zhang Y, Wang Z, Ravid K: The cell cycle in polyploid megakaryocytes is associated with reduced activity of cyclin B1-dependent cdc2 kinase. J Biol Chem 271:4266, 1996. [PMID: 8626773]

75. Hartwig JH: Platelet structure, in *Platelets,* edited by AD Michelson, p 37. Academic Press, San Diego, 2002.
76. Van den Bosch H, de Vet EC, Zomer AW: The role of peroxisomes in ether lipid synthesis. Back to the roots of PAF. *Adv Exp Med Biol* 416:33, 1996.
77. Shuster RC, Rubenstein AJ, Wallace DC: Mitochondrial DNA in anucleate human blood cells. *Biochem Biophys Res Commun* 155:1360, 1988. [PMID: 3178814
78. McNicol A, Israels SJ: Platelet dense granules: Structure, function and implications for haemostasis. *Thromb Res* 95:1, 1999. [PMID: 10403682
79. Harrison P, Cramer EM: Platelet granules. *Blood Rev* 7:52, 1993. [PMID: 8467233]
80. Comfurius P, Bevers EM, Zwaal RFA: The involvement of cytoskeleton in the regulation of transbilayer movement of phospholipids in human blood platelets. *Biochim Biophys Acta* 815:143, 1985. [PMID: 2985115]
81. Escolar G, Krumwiede M, White JG: Organization of the actin cytoskeleton of resting and activated platelets in suspension. *Am J Pathol* 123:86, 1986. [PMID: 2870643]
82. White JG: Anatomy and structural organization of the platelet, in: *Hemostasis and Thrombosis: Basic Principles and Clinical Practice,* edited by RW Colman, J Hirsh, VJ Marder, EW Salzman, p 397. JB Lippincott, Philadelphia, 1993.
83. McEver RP: P-selectin/PSGL-1 and other interactions between platelets, leukocytes, and endothelium, in *Platelets,* edited by AD Michelson, p 139. Academic Press, San Diego, 2002.

84. Loscalzo J, Inbal A, Handin RI: von Willebrand protein facilitates platelet incorporation into polymerizing fibrin. *J Clin Invest* 78:1112, 1986. [PMID: 3489737]
85. Bruschke AV, Sheldon WC, Shirey EK, et al: A half century of selective coronary arteriography. *J Am Coll Cardiol* 2009; 54:2139.
86. Scanlon P, Faxon D, Audet A, et al: ACC/AHA Guidelines for coronary angiography. *J Am Coll Cardiol* 1999; 33:1756.
87. Helft G, Dambrin G, Zaman A, et al: Percutaneous coronary intervention in anticoagulated patients via radial artery access. *Catheter Cardiovasc Interv* 2009; 73:44.
88. Cox N, Resnic FS, Popma JJ, et al: Comparison of the risk of vascular complications associated with femoral and radial access coronary catheterization procedures in obese versus nonobese patients. *Am J Cardiol* 2004; 94:1174
89. *Courtesy of Charles J. Davidson, Northwestern University*
90. Navaneethan S, Singh S, Appasamy S, et al: Sodium bicarbonate therapy for prevention of contrast-induced nephropathy: A systematic review and meta-analysis. *Am J Kidney Dis* 2009; 53:617.
91. Mehran R, Nikolsky E, Kirtane AJ, et al: Ionic low-osmolar versus nonionic iso-osmolar contrast media to obviate worsening nephropathy after angioplasty in chronic renal failure patients: The ICON (Ionic versus non-ionic Contrast to Obviate worsening Nephropathy after angioplasty in chronic renal failure patients) study. *J Am Coll Cardiol Intv* 2009; 2:415
92. *From CASS Principal Investigators and their Associates: Coronary Artery Surgery Study (CASS): A randomized trial of coronary artery surgery: Survival data. Circulation 68:939, 1983*

93. Schwartz L, Kip KE, Alderman E, et al: Baseline coronary angiographic findings in the Bypass Angioplasty Revascularization Investigation 2 Diabetes trial (BARI 2D). Am J Cardiol 2009; 103:632.

94. From Ryan TJ, Bauman WB, Kennedy JW, et al: Guidelines for percutaneous coronary angioplasty. A report of the AHA/ACC Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Percutaneous Transluminal Coronary Angioplasty). Circulation 88:2987, 1993.

95. *Modified from Sheehan F, Braunwald E, Canner P, et al: The effect of intravenous thrombolytic therapy on left ventricular function: A report on the tissue-type plasminogen activator and streptokinase from the Thrombolysis in Myocardial Infarction (TIMI Phase I) trial. Circulation 75:817, 19*

ANNEXURES

IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION

PROFORMA

Name: Patient ID No:

Age/sex: Contact no:

Occupation:

DOA:

DOD:

COMPLAINTS:

- Chestpain
- Palpitation,
- Syncope
- Sweating
- Shortness of breath
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Swelling of legs

PAST HISTORY:

- Diabetes mellitus/Stroke / TIA/Systemic hypertension/Coronary artery disease/Bronchial asthma

PERSONAL HISTORY:

- Smoking
- Alcohol intake

GENERAL EXAMINATION:

VITAL SIGNS:

- Pulse Respiratory Rate:
- Blood pressure Temperature:

SYSTEMIC EXAMINATION:

- CVS:
- RS:
- P/A:
- CNS:

INVESTIGATIONS:

Complete blood count:	**Renal function test-**	**Liver function test:**
WBC:	RBS:	TOTAL BILIRUBIN:
DC: P- L- E-	UREA:	DIRECT:
	CREATININE:	SGOT:
Hb:	SODIUM:	SGPT:
ESR:		ALP:
PLT COUNT:	POTASSIUM:	T.PROTEIN:
		ALBUMIN:

	BEFORE THROMBOLYSIS	AFTER THROMBOLYSIS
MPV		
ECG		
ECHO		

CORONARY ANGIOGRAM:

ABBREVIATIONS

STEMI	-	ST ELEVATION MYOCARDIAL INFARCTION
IHD	-	ISCHEMIC HEART DISEASE
MPV	-	MEAN PLTELET VOLUME
IRA	-	INFARCT RELATED ARTERY
TIMI	-	THROMBOLYSIS IN MYOCARDIAL INFARCTION
LBBB	-	LEFT BUNDLE BRANCH BLOCK
PCI	-	PERCUTANEOUS CORONARY INTERVENTION
CABG	-	CORONARY ARTERY BYPASS GRAFT
SVR	-	SYSTEMIC VASCULAR RESISTANCE
LVEDP	-	LEFT VENTRICULAR END DIASTOLIC PRESSURE
RAAS	-	RENIN ANGIOTENSIN ACTIVATING SYSTEM
ESR	-	ERYTHROCYTE SEDIMENTATION RATE
CRP	-	C-REACTIVE PROTEIN
ESC	-	EUROPEAN SOCIETY OF CARDIOLOGY
ECG	-	ELECTROCARDIOGRAM
ASE	-	AMERICAN SOCIETY OF ECHOCARDIOGRAPHY
AHA	-	AMERICAN HEART ASSOCIATION
LDL	-	LOW DENSITY LIPOPROTEIN
VCAM	-	VASCULAR CELL ADHESION MOLECULE

ICAM	-	INTRACELLULAR ADHESION MOLECULE
MCP	-	MONOCYTE CHEMOATTRACTANT PROTEIN
M-CSF	-	MACROPHAGE COLONY STIMULATING FACTOR
vWF	-	VON WILLEBRAND FACTOR
PSGL	-	P-SELECTIN GLYCOPROTEIN LIGAND
NPH	-	NEUTRAL PROTAMINE HAGEDORN
MRI	-	MAGNETIC RESONANCE IMAGING
EF	-	EJECTION FRACTION
ATP	-	ADENOSINE TRI PHOSPHATE
ADP	-	ADENOSINE DI PHOSPHATE
EDTA	-	ETHYLENEDIAMINE TETRAACETIC ACID
LAD	-	LEFT ANTERIOR DESCENDING ARTERY
RCA	-	RIGHT CORONARY ARTERY
LCX	-	LEFT CIRCUMFLEX ARTERY
SVD	-	SINGLE VESSEL DISEASE
DVD	-	DOUBLE VESSEL DISEASE
TVD	-	TRIPLE VESSEL DISEASE

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Ragavan .K,
Post Graduate in MD Internal Medicine,
Madras Medical College,
Chennai – 600003.

Dear Dr. Ragavan .K,

The Institutional Ethics Committee has considered your request and approved your study titled **"Impact of admission mean platelet volume on the efficacy of thrombolysis in ST elevation myocardial infarction"** No. 37072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

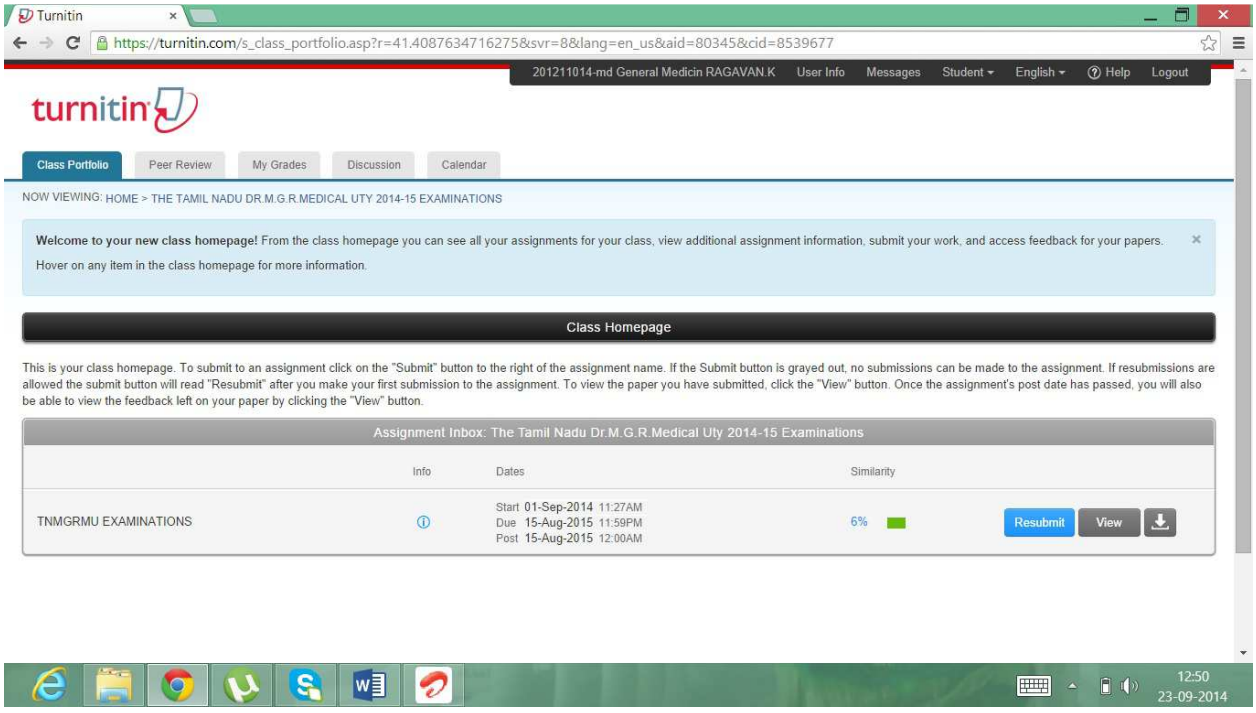
Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Member Secretary, Ethics Committee

TURNITIN PLAGIARISM SCREEN SHOT



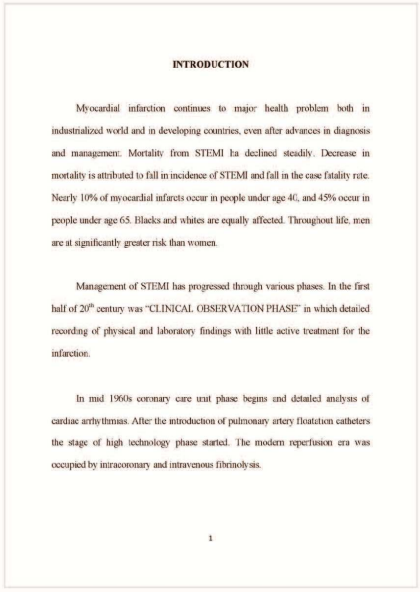
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201211014-md General Medicin RAG..
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	MPV IN STEMI
File name:	Ragavan_thesis.docx
File size:	4.14M
Page count:	115
Word count:	6,502
Character count:	38,388
Submission date:	22-Sep-2014 06:36AM
Submission ID:	453267378



INTRODUCTION

Myocardial infarction continues to major health problem both in industrialized world and in developing countries, even after advances in diagnosis and management. Mortality from STEMI ha declined steadily. Decrease in mortality is attributed to fall in incidence of STEMI and fall in the case fatality rate. Nearly 10% of myocardial infarcts occur in people under age 40, and 45% occur in people under age 65. Blacks and whites are equally affected. Throughout life, men are at significantly greater risk than women.

Management of STEMI has progressed through various phases. In the first half of 20th century was "CLINICAL OBSERVATION PHASE" in which detailed recording of physical and laboratory findings with little active treatment for the infarction.

In mid 1960s coronary care unit phase begins and detailed analysis of cardiac arrhythmias. After the introduction of pulmonary artery floatation catheters the stage of high technology phase started. The modern reperfusion era was occupied by intracoronary and intravenous fibrinolysis.

1

PATIENT CONSENT FORM

Study Detail	:	**"IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION"**
Study Centre	:	RajivGandhiGovernment GeneralHospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (√) these boxes

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

e) I hereby consent to participate in this study. ❑

f) I hereby give permission to undergo detailed clinical examination and blood investigations as required. ❑

Signature/thumb impression	Signature of Investigator
Patient's Name and Address:	Study Investigator's Name: **Dr RAGAVAN K**

INFORMATION SHEET

TITLE:"IMPACT OF ADMISSION MEAN PLATELET VOLUME ON THE EFFICACY OF THROMBOLYSIS IN ST ELEVATION MYOCARDIAL INFARCTION"

NAME OF THE INVESTIGATOR: Dr.RAGAVAN K

STUDY CENTRE: Rajiv Gandhi Government General Hospital, Chennai.

NAME OF THE PARTICIPANT: **AGE:** **SEX:**

PURPOSE OF THE STUDY: To know the impact of admission mean platelet volume on efficacy of thrombolysis in ST Elevation myocardial infarction.

STUDY DESIGN: Observational study

STUDY PROCEDURE: We are selecting certain patients if you are eligible after filling up the questionnaire, 3ml blood will be taken at the time of admission, ECG, blood biochemistry testing will be done. After thrombolysis coronary angiogram will be done. These tests and special studies do not affect your final report or management.

POSSIBLE RISKS: No possible risks by means of this study.

POSSIBLE BENEFITS: if we found there is impact of admission mean platelet volume on efficacy of thrombolysis in ST Elevation myocardial infarction, the success of thrombolysis can be predicted on admission itself.

CONFIDENTIALITY OF THE INFORMATION OBTAINED FROM THE PATIENT: The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

DECISION TO PARTICIPATE IN THE STUDY: Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

RESULT OF THE STUDY:

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator | Signature of Participant

Date :

Place :

MASTER CHART

S.NO.	AGE	SEX	IP.NO	DM	SHT	Smoking	Alcohol	TIME (hours)	Platelet count	MPV		TOTAL COUNT	TYPE OF MI	IRA	Number of vessels	TIMI FLOW	PULSE (per min)	SBP (mm Hg)	DBP (mm Hg)	EF
										BT	AT									
1	62	M	69699	3 Y	3 Y	N	N	6	120000	9.5	9.3	11000	EAWMI	PROXIMAL LAD LCX	DVD	I	90	150	90	40
2	56	M	88532	N	3 Y	N	N	6	84000	11.5	10.5	12500	IWMI	PROXIMAL RCA	SINGLE VESSEL	I	60	90	60	50
3	43	F	89273	M	6 Y	N	N	3	252000	8.9	8.9	9000	IWMI	DISTAL RCA	SINGLE VESSEL	III	65	100	60	60
4	78	M	90883	N	N	Y	Y	5	164000	10.6	10	11200	EAWMI	PROXIMAL LAD LCX	DVD	III	90	110	60	47
5	67	M	69395	6 Y	N	Y	Y	4	156000	9	8.8	7500	AWMI	PROXIMAL LAD	SVD	III	70	110	70	60
6	65	M	82313	5 Y	N	Y	Y	6	120000	10.5	10.3	11000	IWMI + RVMI	PROXIMAL RCA	SVD	I	62	100	50	40
7	58	M	80397	N	N	N	N	6	90000	10.3	10.1	21000	EAWMI	PROXIMAL LAD LCX	DVD	I	100	100	60	45

8	43	M	76682	6 M	N	N	N	3	330000	8.6	8.4	8500	IWMI	DISTAL RCA	SVD	III	70	100	60	64
9	52	F	83722	3 Y	N	N	N	2	120000	8.5	8.5	7500	LWMI	LCX	SVD	III	90	130	70	60
10	38	M	87152	N	N	Y	Y	5.5	100000	10.5	10.5	21100	EAWMI	PROXIMAL LAD RCA	DVD	II	120	110	70	40
11	51	M	90201	N	N	N	N	5.5	150000	10	9.5	13000	EAWMI	PROXIMAL LAD LCX	DVD	I	98	100	60	42
12	45	M	70825	N	N	N	N	2	250000	8	7.8	6200	IWMI	DISTAL RCA	DVD	III	72	100	60	63
13	60	M	76757	10 Y	N	N	N	6	120000	9.8	9.8	13000	AWMI	PROXIMAL LAD LCX	DVD	I	102	100	70	46
14	49	F	89711	-	-	-	-	5	150000	9.5	9	9000	AWMI	PROXIMAL LAD	SVD	II	94	130	60	50
15	56	M	75028	10 Y	8 Y	N	N	6	130000	10.2	10	11000	EAWMI	PROXIMAL LAD MID LCX MID RCA	TVD	II	88	100	70	48
16	48	F	84114	N	N	N	N	3	100000	8.5	8.5	7000	LWMI	LCX	SVD	III	70	110	80	60
17	53	M	77266	N	N	N	N	4	240000	8.5	8	7000	AWMI	DISTAL LAD	SVD	III	74	130	70	64
18	48	M	72576	5 Y	5 Y	Y	Y	6	90000	10.2	10.2	20000	EAWMI	PROXIMAL LAD DISTAL LCX	DVD	I	90	100	60	44
19	56	M	73744	N	N	Y	Y	5	120000	9.2	9	17000	EAWMI	PROXIMAL LAD PROXIMAL LCX	DVD	II	102	130	70	50
20	58	M	78915	N	N	Y	Y	4	240000	8.5	8	9000	AWMI	PROXIMAL LAD	SVD	II	90	100	70	55

21	46	M	76011	N	N	Y	Y	5.5	200000	9	8.8	8000	AWMI	PROXIMAL LAD	SVD	II	88	110	60	56
22	50	M	71738	3 Y	3 Y	N	N	3	210000	8.5	8.5	7000	AWMI	PROXIMAL LAD	SVD	II	92	124	60	55
23	62	F	85647	3 Y	3 Y	N	N	3	110000	8	8	6700	LWMI	LCX	SVD	III	84	100	70	60
24	75	M	79033	20 Y	15 Y	Y	Y	7	90000	12	11.5	20000	EAWMI	PROXIMAL LAD PROXIMAL LCX DISTAL RCA	TVD	I	90	100	50	30
25	55	F	81562	5 Y	5 Y	N	N	6	130000	10.5	10	17000	EAWMI	PROXIMAL LAD + PROXIMAL LCX	DVD	II	94	130	70	40
26	45	F	87357	N	N	N	N	2	300000	8	8	6000	IWMI	DISTAL RCA	SVD	III	66	90	60	60
27	66	M	81542	15Y	10	N	N	5	270000	9.5	9.2	8000	AWMI	PROXIMAL LAD	SVD	II	80	100	70	58
28	30	F	85392	N	N	N	N	4	170000	9.3	9	10000	AWMI	PROXIMAL LAD	SVD	II	96	140	70	58
29	65	M	75277	15 Y	15 Y	N	N	3	250000	8.2	8	7200	IWMI	DISTAL RCA	SVD	II	78	100	60	60
30	55	F	81562	N	N	N	N	3	300000	7.8	7.5	6200	AWMI	DISTAL LAD	SVD	III	84	110	70	62
31	53	M	80268	5 Y	N	N	N	5.5	150000	10.4	10.2	12000	EAWMI	PROXIMAL LAD LCX	DVD	I	94	100	50	48
32	48	M	80852	N	N	Y	Y	6	100000	11.2	11	19000	IWMI + RVMI	PROXIMAL RCA LCX	DVD	I	56	90	50	40
33	57	M	79250	N	N	N	N	4	320000	8.4	8	7000	AWMI	DISTAL LAD	SVD	II	98	130	70	55

34	52	M	77728	5 Y	N	Y	Y	7	90000	12.2	11.8	19000	EAWMI	PROXIMAL LAD + PROXIMAL LCX DISTAL RCA	TVD	I	102	100	50	28
35	52	F	85174	5 Y	3 Y	N	N	5.5	280000	11.4	11	8000	EAWMI	PROXIMAL LAD DISTAL LCX	DVD	II	98	130	70	45
36	60	F	85186	5 Y	2 Y	N	N	6	25000	11	10	9000	IWMI + RVMI	PROXIMAL RCA	SVD	II	64	100	60	50
37	32	M	76822	N	N	Y	Y	6	120000	11.8	11.4	17000	EAWMI	PROXIMAL LAD PROXIMAL LCX	DVD	II	104	120	60	42
38	48	F	76011	N	N	N	N	5	320000	9	8.8	13000	IWMI	PROXIMAL RCA	SVD	III	72	100	60	64
39	43	M	76682	N	N	N	N	4	300000	8.2	8	17000	AWMI	PROXIMAL LAD	SVD	III	90	120	70	60
40	59	F	89619	5Y	5Y	N	N	5.5	120000	10.2	10	13000	LWMI IWMI	PROXIMAL RCA PROXIMAL LCX	DVD	II	98	100	60	55